

**ABDOMINAL CLOSURE WITH ANTIBACTERIAL COATED SUTURE  
MATERIALS AND ITS RELATION TO THE INCIDENCE OF POST  
OPERATIVE SUPERFICIAL SURGICAL SITE INFECTION RATES**

**DISSERTATION SUBMITTED TO  
THE TAMILNADU DR.M.G.R. MEDICAL UNIVERSITY**

**CHENNAI**

*In partial fulfilment of  
the requirements for the degree of*

**MASTER OF SURGERY**

**In**

**GENERAL SURGERY**



**DEPARTMENT OF GENERAL SURGERY**

**TIRUNELVELI MEDICAL COLLEGE**

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Date:

Place: Tirunelveli

**Prof. Dr.R.MAHESWARI M.S.,**

Professor and HOD

Department of General Surgery,

Tirunelveli Medical College & Hospital,  
Tirunelveli.

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Date:

Place: Tirunelveli

**Prof. Dr. R. MAHESWARI M.S.,**

Professor and HOD,

Department of General Surgery,

Tirunelveli medical college & Hospital,

Tirunelveli.

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Date:

**Dr. SITHY ATHIYA MUNAVARAH M.D.,**

Place: Tirunelveli

The Dean,

Tirunelveli medical college & Hospital,

Tirunelveli.

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## INSTITUTIONAL RESEARCH ETHICS COMMITTEE

TIRUNELVELI, STATE OF TAMILNADU, SOUTH INDIA PIN 627011  
91-462-2572793-EXT; 91-462-2572944; 91-462-2579785; 91-462-2572611-16  
online@tvmc.ac.in, tirec@tvmc.ac.in, www.tvmc.ac.in

### CERTIFICATE OF REGISTRATION & APPROVAL OF THE TIREC

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DESIGNATION OF PRINCIPAL INVESTIGATOR: POST GRADUATE IN GENERAL SURGERY  
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2. Study Protocol
3. Department Research Committee Approval
4. Patient Information Document and Consent Form in English and Vernacular Language
5. Investigator's Brochure
6. Proposed Methods for Patient Accrual Proposed
7. Curriculum Vitae of the Principal Investigator
8. Insurance /Compensation Policy
9. Investigator's Agreement with Sponsor
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Registrar, TIREC  
Tirunelveli Medical College, Tirunelveli - 627011  
State of Tamilnadu, South India



Dr. V. Ramasubramanian MD DM  
Member Secretary, TIREC  
Tirunelveli Medical College, Tirunelveli - 627011  
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Date:  
Place: Tirunelveli

**Dr. R. KARTHIKEYAN,**  
Postgraduate in General Surgery,  
Tirunelveli Medical College & Hospital,  
Tirunelveli.

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**Dr. R. KARTHIKEYAN,**  
Postgraduate in General Surgery,



Date:

Tirunelveli Medical College,

Place: Tirunelveli

Tirunelveli.

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# INTRODUCTION

## **INTRODUCTION**

Surgical site infection (SSI) is an immense burden on healthcare resources even in the modern era of immaculate sterilization approaches and highly effective antibiotics. An estimated 234 million various surgical procedures, involving skin incisions requiring various types of wound closure techniques, are performed in the world, with the majority resulting in a wound healing by primary intention.

The most widely recognized definition of infection, which is used throughout the United States and Europe, is that devised and adopted by the Centre for Disease Control and Prevention. An SSI is defined as an infection occurring within 30 days of surgery that meets the following criteria: (1) the diagnosis consists of the infection of an anatomic plane by one of the following manifestations: collection, inflammatory signs (pain, edema, tenderness, redness), dehiscence, or positive culture; and (2) classification according to the anatomic plane as follows: superficial incisional SSI, infection of the skin and subcutaneous tissue; deep incisional SSI, infection of the deep soft tissue (fascia and muscles); and organ/space SSI, infection of the organ/space. In this study, SSIs were categorized by the above classifications.

A system of classification for surgical wounds that is based on the degree of microbial contamination was developed by the US National Research Council group in 1964. Four wound classes with an increasing risk of SSI were described: clean, clean-contaminated, contaminated, and dirty. In this study, SSIs were researched based on each of the wound classes.

Skin wounds are at risk of SSI and therefore may lead to increased morbidity, delayed recovery and prolonged hospital stay. The prevalence of SSI in the developed world is variable but reported figures are estimated at around 5%. The development of SSI is a multifactorial phenomenon, which requires a multimodal approach to prevent and treat it in a timely manner to avoid financial, psychological and health-related quality of life consequences. Various predisposing aetiopathological factors for SSI include immunosuppression, nutritional deficiencies, hypoproteinemias, congestive cardiac failure, and hepatic failure, and renal failure, use of steroids, chemotherapy agents, steroids and diabetes mellitus. In additions to these factors, wound contamination, contaminated instruments, surgical technique and sutures used to close skin have also been reported to be responsible for SSI and cosmetic outcomes. The prevention of the SSI by various invasive and non-invasive interventions is the most common measure surgeons and other healthcare professional advocate to tackle the problem of SSI. This includes use of prophylactic antibiotics and various other multimodal approaches already reported in the medical literature.

Triclosan [5-chloro-2-(2, 4-dichlorophenoxy) phenol] is a broad-spectrum bacteriocidal agent that has been used for more than 40 years in various products, such as toothpaste and soaps. Higher concentrations of triclosan work as a bactericide by attacking different structures in the bacterial cytoplasm and cell membrane. At lower concentrations, triclosan acts as bacteriostatic agent, binding to enol-acyl reductase (ENR), a product of the Fab I gene and thus inhibiting fatty acid synthesis. Use of triclosan-coated sutures should theoretically result in the reduction of SSI. Several studies have shown a reduction in the number of bacteria in vitro and also of wound infections in animals.

The aim of this study was to evaluate whether the incidence of SSIs can be reduced when triclosan coated sutures are used for the closure of the fascia, and to evaluate the incidence of SSIs according to each wound classification

AIMS AND

OBJECTIVES

## **AIMS AND OBJECTIVES OF THE STUDY**

### **AIM OF THE STUDY**

To assess abdominal closure with antibacterial coated suture materials and its relation to the incidence of post-operative superficial surgical site infection rates.

### **OBJECTIVES OF THE STUDY**

1. To compare the incidence of superficial SSI in laparotomy incisions closed with coated polyglactin910 suture with triclosan versus incisions closed with coated polyglactin910 suture without triclosan
2. To study the time frame between surgery and development of SSI
3. To determine which bacteria is commonly associated with SSI after laparotomy closure

# MATERIALS AND METHODS



## **MATERIALS AND METHODS**

### **SOURCE OF DATA**

1) The data will be collected from hospital records of surgery performed, post-operative daily progress notes and outpatient folders and telephonic conversations with patients after discharge

2) Type of subject: all patients undergoing emergency laparotomy procedure for any cause.

3) Choosing subjects: number to be studied: 70-divided as 35 in each group. This number was chosen keeping in mind the time restrictions of the study, the feasibility and ease of calculations.

#### **Inclusion criteria:**

- 1) All patients above the age of 18yrs requiring a laparotomy
- 2) All superficial SSI (skin and subcutaneous layer only) developing within a 30 day period post-surgery, as per the traditional definition.

#### **Exclusion criteria:**

- 1) Patients < 18 yrs. of age
- 2) Deep SSI or Organ space SSI

3) Wound infections occurring beyond the 30 day time period post-surgery.

## **METHOD OF COLLECTION OF DATA**

1) The pre-operative data collected will include the patient's demographics, co-morbidities, laparotomy indication, setting (emergency/elective) and class of wound. Intra-operative data will include the method of painting and draping, duration of the surgery, antibiotics received during surgery, intra-operative findings which will help in classifying the wound (eg: biliary contamination)

Post-operative data include development of superficial SSI as per the standardized means of detecting and diagnosing superficial surgical site infections, and if they did, what organism did the wound swab grow, and how many days after laparotomy did they develop the SSI.

2) The study planned is an observational study. All individuals admitted in one surgical unit undergoing laparotomy will have closure of subcutaneous layer with coated polyglactin 910 with triclosan. All individuals undergoing laparotomy in other surgical units will have closure of subcutaneous closure with coated polyglactin 910 without triclosan. These patients will be followed up for a period of one month post-surgery and the above mentioned data will be collected.

3) The superficial SSI rates will be reported as percentages within each group and compared between the groups using t-test for proportion.

The time frame between surgery and development of superficial SSI will be summarized as mean and standard deviation. This will be compared between the two groups using independent sample t-test, if the data is normally distributed.

The commonly observed bacteria in the 2 groups will be listed as number and percentage.

All statistical tests will be considered significant at  $p < 0.05$  level of significance.

# REVIEW OF LITERATURE

## **REVIEW OF LITERATURE**

### **SURGICAL SITE INFECTIONS**

#### **HISTORICAL ASPECTS**

The ancient Egyptians were the first civilization to have trained clinicians to treat physical ailments. Medical papyri, such as the Edwin Smith papyrus (circa 1600 BCE) and the Ebers papyrus (circa 1534 BCE), provided detailed information of management of disease, including wound management with the application of various potions and grease to assist healing.[1, 2]

Hippocrates (Greek physician and surgeon, 460-377 BCE), known as the father of medicine, used vinegar to irrigate open wounds and wrapped dressings around wounds to prevent further injury. His teachings remained unchallenged for centuries.

Galen (Greek surgeon to Roman gladiators, 130-200 CE) was the first to recognize that pus from wounds inflicted by the gladiators heralded healing (pus bonum et laudabile ["good and commendable pus"]).

Unfortunately, Galen's observation was misinterpreted, and the concept of pus pre-empting wound healing persevered well into the 18th century. The link between pus formation and healing was emphasized so strongly that foreign material was introduced into wounds to promote pus formation-suppuratation. The concept of wound healing remained a mystery, as highlighted by the famous saying by Ambroise Paré (French military surgeon, 1510-1590), "I dressed the wound. God healed it." [3]

The scale of wound infections was most evident in times of war. During the American Civil War, erysipelas (necrotizing infection of soft tissue) and tetanus accounted for over 17,000 deaths, according to an anonymous source in 1883. Because compound fractures at the time almost invariably were associated with infection, amputation was the only option, despite a 25-90% risk of amputation stump infection.

Koch (Professor of Hygiene and Microbiology, Berlin, 1843-1910) first recognized the cause of infective foci as secondary to microbial growth in his 19th century postulates. Semmelweis (Austrian obstetrician, 1818-1865) demonstrated a fivefold reduction in puerperal sepsis by hand washing between performing post-mortem examinations and entering the delivery room.

Joseph Lister (Professor of Surgery, London, 1827-1912) and Louis Pasteur (French bacteriologist, 1822-1895) revolutionized the entire concept of wound infection. Lister recognized that antisepsis could prevent infection. [4] In

1867, he placed carbolic acid into open fractures to sterilize the wound and to prevent sepsis and hence the need for amputation. In 1871, Lister began to use carbolic spray in the operating room to reduce contamination. However, the concept of wound suppuration persevered even among eminent surgeons such as John Hunter. [5]

World War I resulted in new types of wounds from high-velocity bullet and shrapnel injuries coupled with contamination by the mud from the trenches. Antoine Depage (Belgian military surgeon, 1862-1925) reintroduced wound debridement and delayed wound closure and relied on microbiological assessment of wound brushings as guidance for the timing of secondary wound closure.[6] Alexander Fleming (microbiologist, London, 1881-1955) performed many of his bacteriologic studies during World War I and is credited with the discovery of penicillin.

As late as the 19th century, aseptic surgery was not routine practice. Sterilization of instruments began in the 1880s as did the wearing of gowns, masks, and gloves. Halsted (Professor of Surgery, Johns Hopkins University, United States, 1852-1922) introduced rubber gloves to his scrub nurse (and future wife) because she was developing skin irritation from the chemicals used to disinfect instruments. The routine use of gloves was introduced by Bloodgood, a student of Halsted.

Penicillin first was used clinically in 1940 by Howard Florey. With the use of antibiotics, a new era in the management of wound infections commenced. Unfortunately, eradication of the infective plague affecting surgical wounds has not ended because of the insurgence of antibiotic-resistant bacterial strains and the nature of more adventurous surgical intervention in immunocompromised patients and in implant surgery.

## PATHOPHYSIOLOGY

Wound healing is a continuum of complex interrelated biologic processes at the molecular level. For descriptive purposes, healing may be divided into the following three phases:

- Inflammatory phase
- Proliferative phase
- Maturation phase

### **Inflammatory phase**

The inflammatory phase commences as soon as tissue integrity is disrupted by injury; this begins the coagulation cascade to limit bleeding. Platelets are the first of the cellular components that aggregate to the wound, and, as a result of their degranulation (platelet reaction), they release several cytokines (or paracrine growth factors). These cytokines include platelet-derived growth factor (PDGF),



insulin like growth factor-1 (IGF-1), epidermal growth factor (EGF), and fibroblast growth factor (FGF).

Serotonin is also released, which, together with histamine (released by mast cells), induces a reversible opening of the junctions between the endothelial cells, allowing the passage of neutrophils and monocytes (which become macrophages) to the site of injury.

This large cellular movement to the injury site is induced by cytokines secreted by the platelets (chemotaxis) and by further chemotactic cytokines secreted by the macrophages themselves once at the site of injury. These include transforming growth factor alpha (TGF- $\alpha$ ) and transforming growth factor beta (TGF- $\beta$ ).

Consequently, an inflammatory exudate that contains red blood cells, neutrophils, macrophages, and plasma proteins, including coagulation cascade proteins and fibrin strands, fills the wound in a matter of hours. Macrophages not only scavenge but they also are central to the wound healing process because of their cytokine secretion.

### **Proliferative phase**

The proliferative phase begins as the cells that migrate to the site of injury, such as fibroblasts, epithelial cells, and vascular endothelial cells, start to proliferate and the cellularity of the wound increases. The cytokines involved in

this phase include FGFs, particularly FGF-2 (previously known as basic FGF), which stimulates angiogenesis and epithelial cell and fibroblast proliferation.

The marginal basal cells at the edge of the wound migrate across the wound, and, within 48 hours, the entire wound is epithelialized. In the depth of the wound, the number of inflammatory cells decreases with the increase in stromal cells, such as fibroblasts and endothelial cells, which, in turn, continue to secrete cytokines. Cellular proliferation continues with the formation of extracellular matrix proteins, including collagen and new capillaries (angiogenesis). This process is variable in length and may last several weeks.

### **Maturation phase**

In the maturation phase, the dominant feature is collagen. The dense bundle of fibers, characteristic of collagen, is the predominant constituent of the scar. Wound contraction occurs to some degree in primary closed wounds but is a pronounced feature in wounds left to close by secondary intention. The cells responsible for wound contraction are called myofibroblasts, which resemble fibroblasts but have cytoplasmic actin filaments responsible for contraction.

The wound continuously undergoes remodeling to try to achieve a state similar to that prior to injury. The wound has 70-80% of its original tensile strength at 3-4 months after operation.

### **ETIOLOGY**

All surgical wounds are contaminated by microbes, but in most cases, infection does not develop because innate host defenses are quite efficient in the elimination of contaminants. A complex interplay between host, microbial, and surgical factors ultimately determines the prevention or establishment of a wound infection

## **Microbiology**

Microbial factors that influence the establishment of a wound infection are the bacterial inoculum, virulence, and the effect of the microenvironment. When these microbial factors are conducive, impaired host defenses set the stage for enacting the chain of events that produce wound infection.

Most SSIs are contaminated by the patient's own endogenous flora, which are present on the skin, mucous membranes, or hollow viscera. The traditional microbial concentration quoted as being highly associated with SSIs is that of bacterial counts higher than 10,000 organisms per gram of tissue (or in the case of burned sites, organisms per cm<sup>2</sup> of wound).<sup>[7]</sup>

The usual pathogens on skin and mucosal surfaces are gram-positive cocci (notably staphylococci); however, gram-negative aerobes and anaerobic bacteria contaminate skin in the groin/perineal areas. The contaminating pathogens in gastrointestinal surgery are the multitude of intrinsic bowel flora, which include

gram-negative bacilli (eg, *Escherichia coli*) and gram-positive microbes, including enterococci and anaerobic organisms.<sup>[8]</sup>

Gram-positive organisms, particularly staphylococci and streptococci, account for most exogenous flora involved in SSIs. Sources of such pathogens include surgical/hospital personnel and intraoperative circumstances, including surgical instruments, articles brought into the operative field, and the operating room air.

The group of bacteria most commonly responsible for SSIs are *Staphylococcus aureus* strains. The emergence of resistant strains has considerably increased the burden of morbidity and mortality associated with wound infections.

Methicillin-resistant *Staphylococcus aureus* (MRSA) is proving to be the scourge of modern-day surgery. Like other strains of *S aureus*, MRSA can colonize the skin and body of an individual without causing sickness, and, in this way, it can be passed on to other individuals unknowingly. Problems arise in the treatment of overt infections with MRSA because antibiotic choice is very limited. MRSA infections appear to be increasing in frequency and are displaying resistance to a wider range of antibiotics.<sup>[9]</sup>

Of particular concern are the vancomycin intermediate *S aureus* (VISA) strains of MRSA. These strains are beginning to develop resistance to vancomycin, which is currently the most effective antibiotic against MRSA. This

new resistance has arisen because another species of bacteria, called enterococci, relatively commonly express vancomycin resistance.

### **Risk factors (other than microbiology)**

Decreased host resistance can be due to systemic factors affecting the patient's healing response, local wound characteristics, or operative characteristics, as follows:

- Systemic factors - Age, malnutrition, hypovolemia, poor tissue perfusion, obesity, diabetes, steroids, and other immunosuppressants
- Wound characteristics - Nonviable tissue in wound, hematoma, foreign material, poor skin preparation (eg, shaving), and preexistent sepsis.
- Operative characteristics - Poor surgical technique; lengthy operation (>2 hours); intraoperative contamination (eg, from infected theater staff and instruments or inadequate theater ventilation), prolonged preoperative stay in the hospital, and hypothermia

The type of procedure is a risk factor. Certain procedures are associated with a higher risk of wound contamination than others. The National Research Council (NRC) of National Academy of science was the first group to devise a classification system based on the estimated degree of bacterial contamination and demonstrated a direct relationship between the risk of infection and the degree of contamination. This classification is useful in estimating the risk of SSI,

predicting the potential pathogens and determining the need of antimicrobial prophylaxis. It divides wounds into 4 classes namely

1. Clean/ class I wounds
2. Clean-contaminated/ class II wounds
3. Contaminated/class III wounds
4. Dirty wounds

<b>Classification</b>	<b>Criteria</b>
Clean	Elective, not emergency, Non-traumatic, Primarily closed; No acute inflammation; No break in technique; Respiratory, Gastrointestinal, Biliary and Genitourinary tracts not entered.
Clean-contaminated	Urgent or emergency case that is otherwise clean; Elective opening of respiratory, gastrointestinal, biliary or genitourinary tract with minimal spillage Not encountering infected urine or bile; Minor technique break.
Contaminated	Non-purulent inflammation; Gross spillage from gastrointestinal tract; Entry into biliary or genitourinary tract in the presence of infected bile or urine; Major break in technique;

	Penetrating trauma <4 hours old; Chronic open wounds to be grafted or covered.
Dirty	Purulent inflammation (e.g. abscess); Preoperative perforation of respiratory, gastrointestinal, biliary or genitourinary tract; Penetrating trauma >4 hours old.

## DEFINITION AND CLASSIFICATION

The most widely recognized definition of infection, which is used throughout the United States and Europe, is that devised and adopted by the Centres for Disease Control and Prevention. An SSI is defined as an infection occurring within 30 days of surgery that meets the following criteria: (1) the diagnosis consists of the infection of an anatomic plane by one of the following manifestations: collection, inflammatory signs (pain, edema, tenderness, and redness), dehiscence, or positive culture. SSIs are classified into incisional SSIs, which can be superficial or deep, and organ/space SSIs, which affect the rest of the body other than the body wall layers. These classifications are defined as follows:

- Superficial incisional SSI - Infection involves only skin and subcutaneous tissue of incision

- Deep incisional SSI - Infection involves deep tissues, such as fascial and muscle layers; this also includes infection involving both superficial and deep incision sites and organ/space SSI draining through incision
- Organ/space SSI - Infection involves any part of the anatomy in organs and spaces other than the incision, which was opened or manipulated during operation

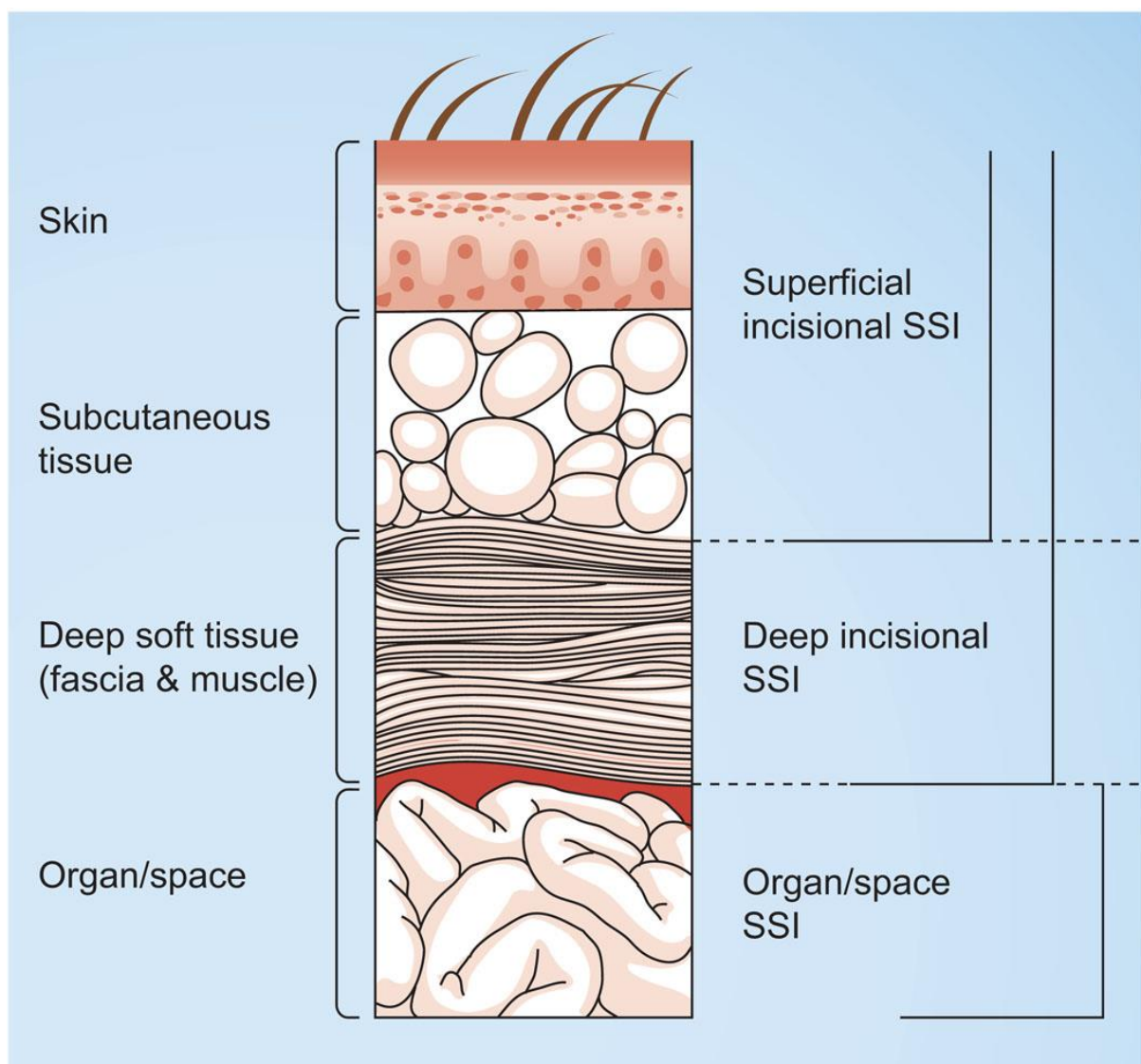


FIGURE 1: CLASSIFICATION OF SURGICAL SITE INFECTIONS



Superficial incisional SSI is more common than deep incisional SSI and organ/space SSI. Superficial incisional SSI accounts for more than half of all SSIs for all categories of surgery. The postoperative length of stay is longer for patients with SSI, even when adjusted for other factors influencing length of stay.

## CRITERIA FOR DEFINING A SURGICAL SITE INFECTION

### SUPERFICIAL INCISIONAL SSI



FIGURE 2: SUPERFICIAL SURGICAL SITE INFECTION

- Infection occurs within 30 days after the operation

- infection involves only skin or subcutaneous tissue of the incision
- *and* at least *one* of the following:
  1. Purulent drainage, with or without laboratory confirmation, from the superficial incision.
  2. Organisms isolated from an aseptically obtained culture of fluid or tissue from the superficial incision.
  3. At least one of the following signs or symptoms of acute inflammation: pain or tenderness, localized swelling, redness, or heat *and* superficial incision is deliberately opened by surgeon, *unless* incision is culture-negative.
  4. Diagnosis of superficial incisional SSI by the surgeon or attending physician.

Do *not* report the following conditions as SSI:

1. Stitch abscess (minimal inflammation and discharge confined to the points of suture penetration).
2. Infection of an episiotomy or new-born circumcision site.
3. Infected burn wound.
4. Incisional SSI that extends into the fascial and muscle layers

#### DEEP INCISIONAL SSI



FIGURE 3: DEEP SURGICAL SITE INFECTION

- Infection occurs within 30 days after the operation if no implant† is left in place or within 1 year if implant is in place and the infection appears to be related to the operation
- infection involves deep soft tissues (e.g., fascial and muscle layers) of the incision *and* at least *one* of the following:
  1. Purulent drainage from the deep incision but not from the organ/space component of the surgical site.
  2. A deep incision spontaneously dehisces or is deliberately opened by a surgeon when the patient has at least one of the following signs of symptoms: fever ( $>38^{\circ}\text{C}$ ), localized pain, or tenderness, unless site is culture-negative.
  3. An abscess or other evidence of infection involving the deep incision is found on direct examination, during reoperation, or by histopathologic or radiologic examination.

4. Diagnosis of a deep incisional SSI by a surgeon or attending physician.

#### ORGAN/SPACE SSI

- Infection occurs within 30 days after the operation if no implant is left in place or within 1 year if implant is in place and the infection appears to be related to the operation
- Infection involves any part of the anatomy (e.g., organs or spaces), other than the incision, which was opened or manipulated during an operation
- *and* at least *one* of the following:
  1. Purulent drainage from a drain that is placed through a stab wound† into the organ/space.
  2. Organisms isolated from an aseptically obtained culture of fluid or tissue in the organ/space.
  3. An abscess or other evidence of infection involving the organ/space that is found on direct examination, during reoperation, or by histopathologic or radiologic examination.
- 4. Diagnosis of an organ/space SSI by a surgeon or attending physician

#### WOUND ASSESSMENT

No validated universal system is designed specifically to aid the assessment and management of surgical wounds. The most commonly used, the CDC definition, employs stringent criteria to classify infection. Several other wound scoring systems exist and two of the best are ASEPSIS and the Southampton Wound Assessment Scale. These enable surgical wound healing to be graded according to specific criteria, usually giving a numerical value, and therefore provide a more objective assessment of the wound.

## ASEPSIS WOUND SCORE

	Proportion of wound affected
Wound characteristic	0 <20 20-39 40-59 60-79
Serous exudate	>80
Erythema	0 1 2 3 4 5
Purulent exudate	0 1 2 3 4 5
Separation of deep tissues	0 2 4 6 8 10
	0 2 4 6 8 10

Points are scored for daily wound inspection.

Criterion	Points
Additional treatment:	
• Antibiotics	10
• Drainage of pus under local anaesthesia	5
• Debridement of wound (general anaesthesia)	10
• Serous discharge*	daily 0 - 5
• Erythema*	daily 0 - 10
• Purulent exudate*	daily 0 - 10
• Separation of deep tissues*	10
• Isolation of bacteria	5
• Stay as inpatient prolonged over 14 days	

\* Given score only on five of seven days. Highest weekly score used

ASEPSIS score = SUM (points from 4 daily wound inspection parameters) + (points for antibiotics) + (points of pus drainage) + (points for wound debridement) + (points for bacterial isolation) + (points for prolonged hospitalization)

Interpretation:

- Minimum score: 0
- maximum score: 70

<b>ASEPSIS score</b>	<b>Interpretation</b>
0 – 10	satisfactory healing
11 – 20	disturbance of healing
21 – 30	minor wound infection
31 – 40	moderate wound infection
> 40	severe wound infection

**TABLE 1: ASEPSIS SCORE GRADING**

Southampton scoring system	
Grade	Appearance
0	Normal healing
I Normal healing with mild bruising or erythema:	
A	Some bruising
B	Considerable bruising
C	Mild erythema
II Erythema plus other signs of inflammation	
A	At one point
B	Around sutures
C	Along wound
D	Around wound
III Clear or haemoserous discharge:	
A	At one point only (<2cm)
B	Along wound (>2cm)
C	Large volume
D	Prolonged (>3 days)
IV Pus:	
A	At one point only (<2cm)
B	Along wound (>2cm)
V Deep or severe wound infection with or without tissue breakdown; haematoma requiring aspiration	

**FIGURE 4: SOUTHAMPTON WOUND GRADING**

Southampton scale - by using the worst wound score recorded and information about any treatment instituted either in hospital or the community, wounds were regarded in four categories:

A. normal healing;

B. minor complication

C. wound infection - wounds graded IV or V, or wounds treated with antibiotics after discharge from hospital, irrespective of the wound grading given to them by the nurse;

D. major hematoma-wound or scrotal hematomas requiring aspiration or evacuation.

## MANAGEMENT OF SURGICAL SITE INFECTIONS

### APPROACH CONSIDERATIONS

Most patients with wound infections are managed in the community. Management usually takes the form of dressing changes to optimize healing, which usually is by secondary intention.

Resultant increased hospital stay due to surgical site infection (SSI) has been estimated at 7-10 days, increasing hospitalization costs by 20%. Occasionally, further intervention in the form of wound debridement and subsequent packing and frequent dressing is necessary to allow healing by secondary intention.



In 2014, the Infectious Diseases Society of America issued the following practice guidelines for the management of SSIs:

Suture removal plus incision and drainage should be performed for SSIs (strong recommendation, low-quality evidence)

Adjunctive systemic antimicrobial therapy is not routinely indicated but, in conjunction with incision and drainage, may be beneficial for SSIs associated with a significant systemic response, such as erythema and induration extending more than 5 cm from the wound edge, temperature exceeding 38.5°C, heart rate higher than 110 beats/min, or white blood cell (WBC) count higher than 12,000/ $\mu$ L (weak recommendation, low-quality evidence)

A brief course of systemic antimicrobial therapy is indicated in patients with SSIs after clean operations on the trunk, head and neck, or extremities that also have systemic signs of infection (strong recommendation, low-quality evidence)

A first-generation cephalosporin or an antistaphylococcal penicillin for methicillin-sensitive *S aureus* (MSSA)—or vancomycin, linezolid, daptomycin, telavancin, or ceftaroline where risk factors for methicillin-resistant *S aureus* (MRSA) are high (nasal colonization, prior MRSA infection, recent hospitalization, or recent antibiotics)—is recommended (strong recommendation, low-quality evidence)

Agents active against gram-negative bacteria and anaerobes, such as a cephalosporin or fluoroquinolone in combination with metronidazole, are recommended for infections after operations on the axilla, gastrointestinal tract, perineum, or female genital tract (strong recommendation, low-quality evidence)

## ANTIBIOTIC PROPHYLAXIS

The use of antibiotics was a milestone in the effort to prevent wound infection. The concept of prophylactic antibiotics was established in the 1960s when experimental data established that antibiotics had to be in the circulatory system at a high enough dose at the time of incision to be effective.

It is generally agreed that prophylactic antibiotics are indicated for clean-contaminated and contaminated wounds. Antibiotics for dirty wounds are part of the treatment because infection is established already. Clean procedures might be an issue of debate. No doubt exists regarding the use of prophylactic antibiotics in clean procedures in which prosthetic devices are inserted; infection in these cases would be disastrous for the patient. However, other clean procedures (eg, breast surgery) may be a matter of contention.

Criteria for the use of systemic preventive antibiotics in surgical procedures are as follows:

Systemic preventive antibiotics should be used in the following cases: A high risk of infection is associated with the procedure (eg, colon resection); consequences

of infection are unusually severe (eg, total joint replacement); the patient has a high NNIS risk index

The antibiotic should be administered preoperatively but as close to the time of the incision as is clinically practical Antibiotics should be administered before induction of anesthesia in most situations

The antibiotic selected should have activity against the pathogens likely to be encountered in the procedure

Postoperative administration of preventive systemic antibiotics beyond 24 hours has not been demonstrated to reduce the risk of SSIs.

Qualities of prophylactic antibiotics include efficacy against predicted bacterial microorganisms most likely to cause infection, good tissue penetration to reach wound involved, cost effectiveness, and minimal disturbance to intrinsic body flora.

The timing of administration is critically important because the concentration of the antibiotic should be at therapeutic levels at the time of incision, during the surgical procedure, and, ideally, for a few hours postoperatively. Antibiotics are administered intravenously, generally 30 minutes prior to incision; they should not be administered more than 2 hours prior to surgery.

Colorectal surgical prophylaxis additionally requires bowel clearance with enemas and oral nonabsorbable antimicrobial agents 1 hour before surgery. High-risk cesarean surgical cases require antibiotic administration as soon as the clamping of the umbilical cord is completed.

## PERIOPERATIVE RECOMMENDATIONS

Perioperative recommendations have been made for minimizing wound infection and SSI, supported by varying degrees of evidence

## PREOPERATIVE PATIENT PREPARATION

Category IA recommendations for preoperative patient preparation include the following:

Identify and treat all infections remote from the surgical site; delay operation in elective cases until infection is treated

Do not remove hair unless it infringes on the surgical field; if hair removal is required, it should be removed immediately before operation and preferably with electric clippers

Category IB recommendations include the following:

Patients should cease tobacco consumption in any form for at least 1 month preoperatively

Optimize blood glucose level and avoid hyperglycemia

Patients are to shower/bathe with antiseptic on at least the night before surgery

Necessary blood products may be administered

The category II recommendation is as follows: Provided that preoperative patient preparation is adequate, minimize preoperative hospital stay.

No recommendations are made regarding the following:

Gradual reduction/discontinuance of steroid use before elective surgery

Enhanced nutritional intake solely to prevent SSI

Preoperative topical antibiotic use in nares to prevent SSI

Measures to enhance wound space oxygenation

## PREOPERATIVE CONSIDERATIONS FOR SURGICAL TEAM MEMBERS

Category IB recommendations regarding preoperative considerations for surgical team members are as follows:

Keep fingernails short; do not wear artificial nails

Scrub hands and forearms as high as the elbows for at least 2-5 minutes with appropriate antiseptic

After scrub, keep hands up with elbows flexed and away from the body; use a sterile towel to dry the hands and put on a sterile gown and gloves

Masks should be worn in the operating suite if sterile instruments are exposed and throughout the surgical procedure; masks should cover the mouth and nose

The hair on the head and face is to be covered with a hood or cap

Liquid-resistant sterile surgical gowns and sterile gloves are to be worn by scrubbed surgical team members

Visibly soiled gowns are to be changed

Shoe covers are not necessary

Routine exclusion of personnel colonized by organisms, such as *S aureus* or group A streptococci, is not necessary unless they are specifically linked to dissemination of such organisms

Personnel with skin lesions that are draining are to be excluded from duty until treated and the infection has resolved

Educate and encourage surgical personnel regarding reporting illness of transmissible nature to supervisory and occupational health personnel

Policies should be established concerning patient care responsibilities for personnel with potentially transmissible infective illnesses, to include aspects of work restrictions, personnel responsibility in utilizing health services, and declaring illness; policies also should direct the responsible person to remove personnel from duty, and policy should be established for clearance to resume work

Category II recommendations are as follows:

Clean under the fingernails prior to the first scrub of the day

Do not wear arm/hand jewelry

No recommendations are made regarding the following:

Nail polish

Restriction of scrub suits to the operating theater

Covering the scrub suits when outside the theater

How or where to launder theater suites

## PREOPERATIVE AND POSTOPERATIVE WOUND CARE

A category IA recommendation for preoperative and postoperative wound care is that asepsis is necessary in the insertion of indwelling catheters, such as intravascular, spinal, or epidural catheters, and subsequent infusion of drugs.

## ABSCCESS SECONDARY TO A SUBCLAVIAN LINE.

Category IB recommendations include the following:

Handle tissues gently with good hemostasis, minimize foreign bodies, and minimize devitalized tissue and dead space

For class III and IV wounds, use delayed closure or leave the wound incision open to heal by secondary intention

If draining of a wound is necessary, the drain exit should be via separate incision distant from the wound; remove the drain as soon as possible

Primary closed incisions should be protected with a sterile dressing for 24-48 hours

Hands are to be washed before and after wound dressing changes/or contact

Category II recommendations include the following:

Use sterile technique for wound dressing change

Educate the patient and relatives regarding wound care symptoms of SSIs and the need to report such problems

Theater environment and care of instrumentation

Category IB recommendations for the theater environment and the care of instrumentation include the following:

Maintain positive-pressure ventilation of the operating suite relative to corridors and surrounding areas

Maintain a minimum of 15 air changes per hour, with at least three being fresh air



Appropriate filters (as recommended by the American Institute of Architects) should be used for filtration of all air, whether recirculated or fresh

Air should enter through the ceiling and exit near the floor

Keep operating room doors closed except for necessary entry

The use of ultraviolet lamps in the theater is not necessary as a deterrent of SSI

Prior to subsequent procedures, visibly soiled surfaces should be cleaned with Environmental Protection Agency (EPA)–approved disinfectants

After a contaminated or dirty procedure, special cleaning or closure of the operating suite is not necessary

Use of tacky mats prior to entry in the operating suite is not necessary

Sterile surgical instruments and solutes should be assembled just prior to use

All surgical instruments should be sterilized according to guidelines; flush sterilization should only be used for instruments that are required for immediate patient use

Category II recommendations include the following:

Limit the number of personnel entering the operating suite.

Orthopedic implant surgery should be performed in an ultraclean-air environment.

Wet-vacuum the floor of the operating theater at the end of day/night using an EPA-approved disinfectant

## SPECIAL SITUATIONS

### ELECTIVE COLON SURGERY

Bowel surgery results in the breakdown of the protective intestinal mucous membrane, with release of the facultative and anaerobic bacteria that heavily colonize the distal small bowel and colon. Eradication of aerobes and anaerobes is necessary to reduce infective complications following intestinal procedures. Mechanical cleansing and antibiotics could achieve this.

Mechanical cleansing can take the form of dietary restrictions; whole gut lavage with one of several preparations, such as 10% mannitol solution, Fleet's phospho-soda, or polyethylene glycol, usually is performed on the day of surgical intervention. Enteral antibiotic regimes to eradicate intrinsic bowel flora vary, with oral neomycin and erythromycin being the most popular combination in the United States. Other combinations with neomycin include the use of metronidazole and tetracycline. Prophylactic parenteral antibiotics also are used with the above.

### INTRAVASCULAR DEVICE-RELATED INFECTIONS

Intravascular devices are of vital use in daily hospital practice. They are used for the parenteral administration of fluids, blood products, nutritional fluids, and

medication and for access in hemodialysis; equally important is their use in the monitoring of critically ill patients.

Unfortunately, because the use of these devices constitutes an invasive procedure, they are associated with infectious complications that could be of a local or systemic nature. Recommendations for prevention and treatment are available to limit their associated morbidity and mortality (which could be as high as 20% in patients with catheter-related bloodstream infections).

In a double-blind, randomized, controlled study of 400 patients with nontunnelled central venous catheters, Dettenkofer et al investigated the effectiveness of the antiseptic octenidine dihydrochloride, used in combination with alcohol-based antiseptic, against infection at central venous catheter insertion sites. One group of patients received skin disinfection with 0.1% octenidine with 30% 1-propanol and 45% 2-propanol, while a control group was disinfected with 74% ethanol with 10% 2-propanol.

In this study, microbial skin colonization at the catheter insertion site and positive microbial cultures at the catheter tip were significantly reduced in the octenidine group. No significant differences in catheter-associated bloodstream infections were found between the groups.

## SURGICAL CARE

Although the goal of every surgeon is to prevent wound infections, they will arise. Treatment is individualized to the patient, the wound, and the nature of the infection. The operating surgeon should be made aware of the possibility of infection in the wound and determine the treatment for the wound.

Ideally, surgical care should start with meticulous detail to strategies that prevent the development of SSIs in the first place. Preoperatively, attention should be paid to factors like optimization of patient status, proper asepsis, and surgical site preparation. Intraoperatively, adherence to good basic surgical principles of minimal and fine tissue dissection, proper selection of suture materials, and proper wound closure is important.

If a SSI sets in, the treatment often involves opening the wound, evacuating pus, and cleansing the wound. The deeper tissues are inspected for integrity and for a deep space infection or source. Dressing changes allow the tissues to granulate, and the wound heals by secondary intention over several weeks. Early/delayed closure of infected wounds is often associated with relapse of infection and wound dehiscence.

## ADDITIONAL PREVENTIVE STRATEGIES

Evidence shows that the close regulation of blood sugar may be a major determinant of wound morbidity. Although investigators have vigorously

pursued for decades the identification of a specific innate or acquired immune deficiency among patients with diabetes, it may be the blood sugar that is the determinant of infection for these patients.

A second issue of considerable interest is body temperature. A prospective randomized study demonstrated that failure to maintain intraoperative core body temperature within 1-1.5°C of normal increases the SSI rate by a factor of 2. It raises the scientific question of whether increasing core temperature during operations over normal temperature might in fact protect against infection.

A third issue is oxygenation. The fresh, hemostatic surgical incision is a hypoxic, ischemic environment. Maintaining or increasing oxygen delivery to the wound by increasing the inspired oxygen concentration administered to the patient perioperatively has also been shown to reduce the incidence of SSIs. It is presumed that increased oxygen availability is a positive host factor, perhaps via enhanced production of oxidant products that facilitate phagocytic eradication of microbes.

Cleaning the wound margins with povidone-iodine before skin closure.

A strategy that could bear fruit for preventing SSIs in the future is the establishment of dedicated infection surveillance units in hospitals with the aim of accomplishing the following:

Identify epidemics by common or uncommon organisms

Establish the correct use of prophylaxis (ie, timing, dose, duration, choice)

Document costs, risk factors, and readmission rates

Monitor postdischarge infections and secondary consequences

Ensure patient safety

A major concern is how to prevent or minimize the emergence of resistance. Although resistance is not a new phenomenon, the incidence has increased dramatically over the past two decades. The development of new drugs has slowed considerably and may be unable to keep pace with the continuing growth of pathogen resistance.

Accordingly, effective strategies are needed to prevent the continuing emergence of antimicrobial resistance. These strategies include avoiding unnecessary antibiotic administration and increasing the effectiveness of prescribed antibiotics, as well as implementing improvements in infection control and optimizing medical practice.

Although an SSI rate of zero may not be achievable, continued progress in understanding the biology of infection at the surgical site and consistent applications of proven methods of prevention will further reduce the frequency, cost, and morbidity associated with SSIs.

## TRICLOSAN

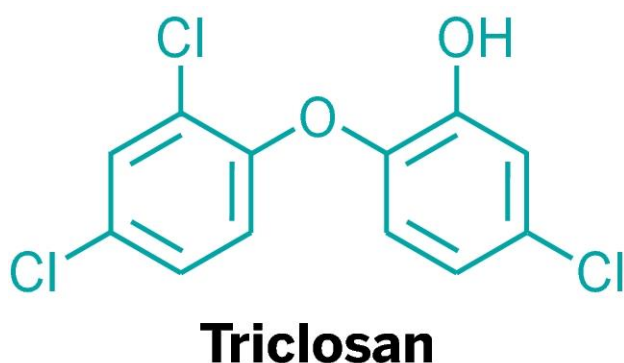


FIGURE 5: TRICLOSAN MOLECULAR STRUCTURE

Triclosan [5-Chloro-2-(2, 4-dichlorophenoxy) phenol] is a broad-spectrum antibacterial agent that inhibits bacterial fatty acid synthesis at the enoyl-acyl carrier protein reductase (FabI) step. Resistance to triclosan in *Escherichia coli* is acquired through a missense mutation in the *fabI* gene that leads to the expression of FabI [G93V]. The specific activity and substrate affinities of FabI [G93V] are similar to FabI. Two different binding assays establish that triclosan dramatically increases the affinity of FabI for  $\text{NAD}^+$ . In contrast, triclosan does not increase the binding of  $\text{NAD}^+$  to FabI [G93V]. The x-ray crystal structure of the FabI- $\text{NAD}^+$ -triclosan complex confirms that hydrogen bonds and hydrophobic interactions between triclosan and both the protein and the  $\text{NAD}^+$  cofactor contribute to the formation of a stable ternary complex, with the drug binding at the enoyl substrate site. These data show that the formation of a noncovalent “bi-substrate” complex accounts for the effectiveness of triclosan as a FabI inhibitor and illustrates that mutations in the FabI active site that interfere with the

formation of a stable FabI-NAD<sup>+</sup>-triclosan ternary complex acquire resistance to the drug.

The fatty acid synthase system of *Escherichia coli* is the paradigm for the type II or dissociated fatty acid synthase systems. Distinct genes encode each of the individual enzymes, and the same basic chemical reaction is often catalyzed by multiple isozymes. There are four basic reactions that constitute a single round of elongation. The first step is the condensation of malonyl-ACP<sup>1</sup> with either acetyl-CoA to initiate fatty acid synthesis (FabH) or with the growing acyl chain to continue cycles of elongation (FabB or FabF). The  $\beta$ -ketoacyl-ACP is reduced by an NADPH-dependent  $\beta$ -ketoacyl-ACP reductase (FabG). Only a single enzyme is responsible for this step. There are two  $\beta$ -hydroxyacyl-ACP dehydrases (FabA and FabZ) capable of forming *trans*-2-enoyl-ACP. The product of the *fabA* gene is specifically involved in the introduction of a *cis* double bond into the growing acyl chain at the  $\beta$ -hydroxydecanoyl-ACP step and most efficiently catalyzes dehydration of short-chain  $\beta$ -hydroxyacyl-ACPs, whereas the FabZ dehydratase has a broader substrate specificity. The last reaction in each elongation cycle is catalyzed by enoyl-ACP reductase (FabI). Contrary to the initial conclusion that there were two enoyl-ACP reductases, based on assays in crude extracts, *E. coli* cells possess only a single NADH-dependent enoyl-ACP reductases encoded by the *fabI* gene that utilizes all chain lengths.



The importance of fatty acid biosynthesis to cell growth and function makes this pathway an attractive target for the development of antibacterial agents. Two important control points in the cycle are the condensing enzymes and the enoyl-ACP reductase, and both reactions are targeted by compounds that effectively inhibit fatty acid synthesis. Two natural products, cerulenin and thiolactomycin, are potent antibiotics that function by specifically inhibiting the condensing enzyme reactions. The diazaborines, a class of heterocyclic antibacterials, inhibit fatty acid biosynthesis by blocking the FabI step. Resistance to the diazaborines arises from a missense mutation in the *fabI* gene that leads to the expression of a FabI [G93S] mutant protein. Similarly, the *fabI* analog in *Mycobacterium tuberculosis*, the *inhA* gene, encodes a cellular target for isoniazid and ethionamide. A point mutation in the *inhA* gene confers resistance to the drugs. Both isoniazid and diazaborine bind at the substrate site of the respective enoyl-ACP reductases and covalently react with NAD<sup>+</sup> to form tight binding bi-substrate complexes. Triclosan is a broad-spectrum antibacterial agent that enjoys widespread applications in a multitude of contemporary consumer products including, soaps, detergents, toothpastes, skin care products, cutting boards, and mattress pads. Triclosan is widely thought to be a nonspecific biocide that attacks bacterial membranes, and if triclosan does not have a discrete mechanism of action, the acquisition of cellular resistance is unlikely. However, recent work reveals that resistant *E. coli* strains arise from missense mutations in

the *fabI* gene and that triclosan and other 2-hydroxydiphenyl ethers directly inhibit fatty acid biosynthesis *in vivo* and FabI catalysis *in vitro*.

## SAFETY

Triclosan passively dissipates from implanted sutures to the surrounding tissues where it is absorbed into the bloodstream and widely distributed, but not confined to any particular tissue or organ system. Triclosan is rapidly metabolised in the liver principally by Phase II metabolism to glucuronide and sulphate conjugates with an elimination half-life of 13 hours after a single oral exposure (11). Therefore, triclosan is cleared from the bloodstream (over 99%) in approximately 3–8 days. Conjugated triclosan is readily water-soluble and is excreted from the body by the kidneys. There is no evidence that triclosan accumulates in the body over time and this pharmacokinetic profile makes it suitable for clinical use.

Selected pharmacokinetic parameters after oral exposure to triclosan were compared between humans and hamsters to determine the usefulness of hamsters to simulate a human pharmacokinetic response (12). Triclosan was well-absorbed after oral administration in both species. The predominant metabolite was the glucuronide conjugate of triclosan. The elimination half-life was 11–20 hours in humans compared with 24–32 hours in hamsters, indicating a more rapid elimination of triclosan for humans. The major route of excretion was via the kidneys for both species. Overall, the hamster is considered to be a good surrogate

for humans with respect to the absorption, distribution, metabolism and elimination of triclosan.

Although the pharmacokinetic studies with triclosan have been conducted principally after oral or topical routes of exposure (13), some intravenous studies in animals have been conducted to determine absolute bioavailability (14). Intravenous exposure by-passes the possibility of first-pass metabolism and is considered to represent a worst-case of what would happen after implantation of a suture. Comparing results for hamsters and humans, a similar pattern of metabolism was observed with the predominant metabolite being the glucuronide conjugate with free triclosan found in the urine. A similar pattern of elimination was also observed with >90% radiolabel being excreted in 7 days with <1% being found in major organs/tissues and no evidence of accumulation in the body. Overall, the similar metabolic pathway of triclosan after intravenous exposure allows for the use of the extensive safety database available after oral exposure to support the safety of Vicryl Plus (Ethicon, Inc., Somerville, NJ) suture.

There is no associated experimental chronic or major adverse target organ toxicity, carcinogenicity, or potential for mutagenic, clastogenic or teratogenic effects and no adverse effects on male or female fertility, or endocrine function. World-wide topical exposure to triclosan-containing personal care products indicates that the sensitisation potential of triclosan is low.

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## Assessment of patient exposure

Maximal single-day patient exposure to triclosan has been determined by using the in vivo dissipation rate from sutures to calculate margins of safety for systemic toxicity. For triclosan coated (Vicryl Plus) suture, 69% of the total triclosan content dissipates in the first 24 hours after implantation, with 99% dissipation by 36 days. Monocryl Plus (coated) and PDS Plus (impregnated) have their own dissipation profiles (15).

### · Potential for systemic toxicity

Assuming the intra-operative, ‘worst-case’ use of 5 m of a 2–0 suture with 472 µg triclosan/m for Vicryl Plus and 2360 µg/m for Monocryl Plus and PDS Plus, and considering the specific dissipation profile of triclosan from each suture, the maximal single-day exposure to triclosan was calculated to be 0 · 03, 0 · 08 and 0 · 09 mg/kg body weight, respectively. Margins of safety, calculated by dividing the No-Observed-Effect-Levels from systemic toxicity studies by the maximal single-day exposure, range from 140 to 2500 (Tables 2 and 3) (16). Margins of safety of 100 are considered sufficient to ensure the safety of many substances. When compared to the widespread use of triclosan-containing oral and topical personal care products, the contribution of a maximal single daily exposure to triclosan from Vicryl Plus is only 12% of daily background exposure. Similarly, a maximal single daily exposure to triclosan from Monocryl Plus and

PDS Plus is 33% and 37%, respectively, compared to daily exposure from combined personal care products.

- Local irritant potential

Clinically relevant intradermal injections of Plus sutures result in a negligible irritant response. Studies of intramuscularly implanted Plus sutures showed that the tissue reaction, absorption profile and impact on wound healing at the implantation site were comparable to that observed for control sutures not containing triclosan.

- Impact on wound healing

Segments of Plus sutures placed in experimental incisional skin wounds caused no adverse cosmetic effects or changes in multiaxial biomechanical wound strength over time.

Aside of multiple clinical studies discussed later, which did not report any interaction with wound healing; only one randomised prospective pilot study (16) reported that triclosan-coated sutures seem to have adverse effects on wound healing. The authors investigated the effect of a triclosan-coated suture on wound healing in 26 women undergoing a breast reduction in comparison to a similar suture without triclosan-coating. The main outcome measure was the incidence of wound dehiscence. In breasts operated on with triclosan-sutures, there was a wound dehiscence in 16 cases, whereas in the control group without triclosan

dehiscence only occurred in seven cases ( $P = 0.023$ ). The authors explained the difference in the two groups by formation of chloroform and other chlorinated daughter products as a reaction of triclosan with free chlorine interacting with wound healing.

However, the required amount of free chlorine, optimum pH, temperature and ultra violet radiation required for this reaction, are not present in the post-surgical scar tissue and patients' subcutaneous tissue, making it difficult to conclude that triclosan-coated surgical sutures are a cause for wound dehiscence.

- Impact on reduction of infection



FIGURE 6: Bacterial colonies (dark red) are visible in all areas of a petri dish except for the "zone of inhibition" around an Antibacterial Suture

Treating sutures with triclosan provides an effective strategy for reducing SSIs because bacterial contamination of suture material within a surgical wound may increase the virulence of a SSI (17). Numerous studies have confirmed the utility of these sutures in decreasing both bacterial colonisation of sutures and wound infections after surgery (18).

## TRICLOSAN AND THE RISK OF RESISTANCE

Bacteria have evolved to survive natural and man-made stresses but there is no evidence of adverse effects of resistance caused by triclosan in the environment, even after long-term exposure (19). Therefore, there is apparently a disparity between what can be shown in laboratory studies and what happens in the real world environment for this molecule. For example, the oral cavity represents an environment that may be commonly exposed to triclosan but triclosan has been safely used in dental hygiene for some 40 years without evidence of dysbiosis, resistance or cross-resistance.

The same terminology of 'resistance' is frequently used for antiseptics and antibiotics, often incorrectly. The term insusceptibility normally refers to resistance because of innate physiological properties of a bacterium: for example, many 'environmental' bacteria are not susceptible to a wide range of antimicrobials, including antiseptics, antibiotics and disinfectants. Resistance is

a clinical term which describes a change in susceptibility that may result in failure of a treatment with an antibiotic.

Antiseptics are in many respects distinct from antibiotics. They were described by Hippocrates and the ancient Egyptians, and have been in widespread clinical use for the last 50–100 years. Insusceptibility has been noted to some antiseptics and is based on alterations in bacterial physiology (bacterial cell walls, membrane proteins and efflux pumps, cytoplasmic organelles and cell respiratory processes, enzymes and nucleic acid) that may or may not be reversible. However, smaller changes in susceptibility have also been noted to a wide variety of antiseptics and can be reproduced in the laboratory for some combinations of bacteria and antiseptics. The occurrence and implications of antibiotic resistance are well known, but less so for antiseptics.

Changes in susceptibility can be shown in the laboratory to some agents, including triclosan, but this is not universal to all organisms and to date this has not been shown clinically, or environmentally, for triclosan. Cross-resistance (where exposure to an antiseptic causes antibiotic resistance) has also not been conclusively showed for triclosan in the clinical, or other environments. The widely accepted and unambiguous cause of antibiotic resistance is the use and misuse of antibiotics. Antibiotics often have a single or limited number of pharmacological targets in microorganisms, whereas antiseptics generally have multiple targets, depending on concentration. Antiseptics have a long history of



use, with early examples being Semmelweis' chlorinated lime solutions (1848) and Lister's carbolic spray (1869). True antiseptic 'resistance' is not frequently encountered and outcome altering changes in susceptibility are uncommon.

Antibiotic resistance is defined as a change from a susceptible phenotype to a less susceptible phenotype which results in clinical, therapeutic failure. In the case of antiseptics, since commonly 100 times higher concentrations of antiseptic are used than is needed, a fourfold decrease in bacterial susceptibility will not result in therapeutic failure and is therefore not resistance in the true sense of the word. The number of cellular mechanisms, disrupted by antiseptics, increases with increasing concentration and, at the high concentrations used in practice to achieve rapid micro biocidal action, antiseptics generally produce many potentially lethal effects on the bacterium, such as disruption of the cell membrane or inactivation of a enzymes, dissipation of transmembrane ion gradients, etc. At lower, growth inhibiting concentrations they may act in the same way as antibiotics, specifically affecting one or two cellular targets. However, even for antiseptics that affect multiple targets, the susceptibility of each target is likely to be variable and dependent on the concentration of the antiseptic (20).

	MIC	MBC	MBEC
<i>Pseudomonas aeruginosa</i>	900	1000	1200
<i>Enterococcus faecalis</i>	4	4	50
<i>Escherichia coli</i>	<1	<1	200
<i>Stenotrophomonas maltophilia</i>	15	60	600
<i>Klebsiella pneumoniae</i>	<1	30	150
<i>Corynebacterium xerosis</i>	10	10	20
<i>Micrococcus luteus</i>	2.0	9	50
<i>Staphylococcus haemolyticus</i>	<1	7.0	30
<i>Staphylococcus aureus</i>	<1	2	34
<i>Candida albicans</i>	10	24	–

TABLE 2: ANTIBIOTIC SPECTRUM OF TRICLOSAN

## TRICLOSAN COATED SUTURES

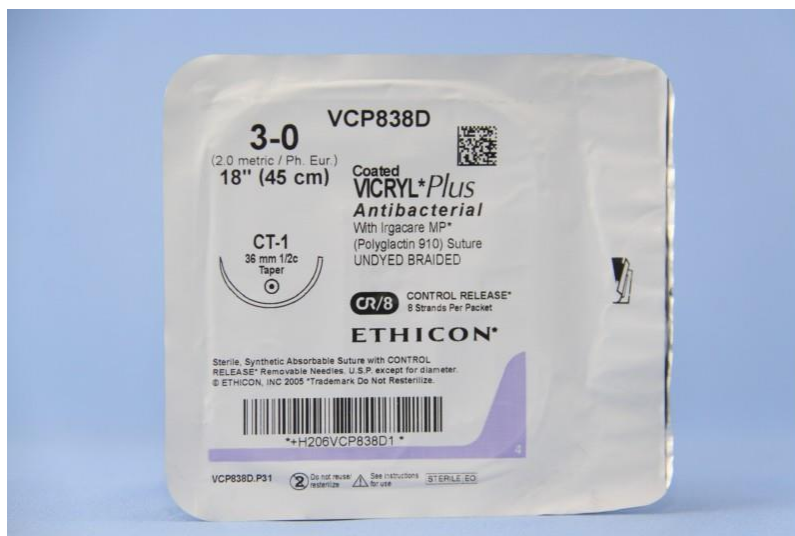


FIGURE 7: TRICLOSAN COATED SUTURE

Suture material is known to be a potential agent of infection [21). To prevent microbial colonization of suture material in operative wounds, Triclosan-coated polyglactin 910 suture materials with antibacterial activity (Vicryl Plus

and Monocryl plus Ethicon GmbH, Nordersdedt, Germany) have been developed. Triclosan (TC) is a broad-spectrum phenol family antiseptic, used for more than 30 years as a safe and effective antimicrobial agent [22], against the most common pathogen agents that cause SSI: *S. aureus* and *S. epidermidis*. The antimicrobial efficacy of this material in reducing both bacterial adherence to the suture and microbial viability have been proven in vitro [23, 24] and in animal models [25–26]. Coated sutures with TC were compared clinically to no impregnated suture material in extragynecological surgery, and were shown to perform as well or better than traditional sutures with respect to intraoperative handling and wound healing in pediatric general surgery, pediatric neurosurgery, thoracic, and abdominal surgery. However, other studies suggest that TC-coated sutures could be inefficient or might have potential adverse effects as wound dehiscence, and should be used with caution [27, 28].

A study by Edmiston CE, Seabrook GR et al titled “Bacterial adherence to surgical sutures: Can antibacterial coated sutures reduce microbial contamination?” published in J Am Coll Surg 2006 proved that treating polyglactin910 with triclosan was an effective strategy in decreasing SSI by proving decreased adherence of both Gram positive and Gram negative bacteria to Triclosan coated suture material.

The addition of Triclosan to polyglactin910 suture does not affect the physical handling properties or performance characteristics like the ease of passage through tissues, first throw knot holding, knot security and so on. [39]

Finally, and most importantly, the significant decrease in wound infection rates in patients in whom coated polyglactin910 with triclosan was used in closure of abdominal wounds was demonstrated by *Justinger C, MoussavianMR, SchleveterC et al and published in Surgery-antibacterial coating of abdominal closure sutures and wound infection 2009.[40]*

The use of triclosan +coated polyglactin910 in subcutaneous closure, by inhibiting bacterial colonization of the suture decreased pain, which can be used as an indicator of subclinical infection was proven in a study conducted by *FordHR, JonesP, GainesB, ReblockH et al and published in Surg Infections (Larchmt) 2005-Intra-operative handling and wound healing: controlled clinical trial comparing coated polyglactin910 with triclosan with coated polyglactin910 suture. [41]*

Another study done to appraise the efficacy of coated Vicryl plus Antibacterial suture (coated polyglactin 910 with Triclosan) in two animal models of general surgery studied the inflammatory and wound healing processes. The results concluded that the antiseptic coated sutures normalize the wound healing process and had an anti-inflammatory effect. [42]

A prospective, randomized, double- blinded controlled multicenter study was done to evaluate the impact of using triclosan- antibacterial sutures on incidence of surgical site infection. It was observed the incidence of surgical site infection was 7% in the study group compared to 15% in the control group and hence concluded that use of triclosan – coated polyglactin 910 antimicrobial suture lead to reduction of surgical site infection. [43]

A study by Ming ET all showed that Poliglecaprone 25 suture with triclosan inhibited bacterial colonization of the suture compared with untreated suture after direct in vivo challenge with *S. aureus* and *E. coli* in animal models. [44]

Another study revealed that Triclosan reduced in vitro colonization of poliglecaprone 25 suture by several strains of bacteria compared with untreated control sutures. [45]

A study showed that The in vitro model demonstrated a considerable reduction ( $p < 0.01$ ) in Gram-positive and Gram-negative bacterial adherence to a triclosan-coated braided suture, which was associated with decreased microbial viability ( $p < 0.001$ ). Because bacterial contamination of suture material within a surgical wound may increase the virulence of a surgical site infection, treating the suture with triclosan provides an effective strategy for reducing perioperative surgical morbidity. [46]

Studies have shown use of these suture (monocryl plus) in decreasing both bacterial colonisation of suture and infections after surgery. Many studies support the hypothesis that triclosan can reduce the risk of suture-associated surgical site infections. [47]

A recent single centre prospective double blinded randomised control trial<sup>21</sup> done in the United States of America looked at the use of triclosan coated sutures (polyglactin 910) inclosure of galea and fascia in cerebrospinal fluid shunts surgery and infection rates. The results were: the incidence of infection in the study group was 2(4.3%) of 46 while in the control 8(21%) of 38. The study was halted prematurely by the researchers after they realised significantly higher infection rates in the control group. The conclusion drawn from that study was that antimicrobial sutures was associated with a reduced risk of postoperative shunt infection, however the study was terminated prematurely at 6 months and it recommended further studies be done to confirm this association.[48]

A double blinded randomised control trial done in Thammasat university Thailand, evaluated the efficacy and safety of vicryl plus compared to vicryl in reducing surgical site infection in appendectomy operation. In the study either vicryl plus or vicryl was used to close the abdominal sheath and the patients were followed up for one year. The preliminary results showed that there was no statistical difference in the surgical site infection between vicryl and vicryl plus (8 and 10%,  $p=0.05$ ). [49]

In another study done in Japan looked at the use of triclosan coated sutures in colorectal surgery. All the patients received intravenous antibiotics pre and post operatively. In the study they also included patients with diabetes mellitus, smokers and on steroids. The infection rate was 4.3% for the vicryl plus group while 9.3% for the control. There was a statistically significant difference in the two groups. The conclusion from the study was that triclosan coated sutures can reduce the incidence of wound infection in colorectal surgery. [50]

We believe that this study presented an achievement by confirming that the use of triclosan-coated sutures (Vicryl Plus) in midline laparotomy can reduce SSIs. Triclosan-coated sutures may be one of the most effective antimicrobial agents developed to date and they are expected to greatly contribute to decreasing SSIs if combined with refined and aseptic surgery Techniques and the proper use of prophylactic antibiotics. SSI rates using triclosan-coated sutures were greatly decreased to 1.39% in gastric cancer patients, compared with the historical data. [51]

SRC rates were 13% and 8%, respectively, for Group 1 and Group 2, which is consistent with most studies (0.8–45%) [29–31]. The observed complication rate for patients treated with TC-coated suture material seemed to be similar to that observed during the first study period (Table 2) and may be wrongly interpreted as unsatisfactory. However, based on patient and surgery characteristics, the complication rate was expected to be higher. This was shown

through the use of a model that was based and validated on the classical population. [52]

In the control group, 75 patients (12.2%) developed wound infections. In the study group, 31 patients (6.6%) developed wound infections, which was significantly lower. Emergency cases; laparoscopic cases, including some cholecystectomy and colectomy cases; American Society of Anaesthesiologists classification; the use of immunosuppressive therapy; colostomy cases; wound classification; and suture material were identified as the risk factors for wound infections. In both groups, as the wound classification worsened, the wound infection rate increased. Triclosan-coated polyglactin 910 antimicrobial sutures lead to a significant decrease in the incidence of surgical site infections, especially in clean/contaminated cases. [53]

When a PDS loop suture for abdominal wall closure was used, 42 (11.3%) patients with wound infections were detected. The number of patients with wound infections decreased significantly to 31 when the PDS plus for abdominal wall closure was used (6.4%,  $P < .05$ ). Other risk factors for the development of side infections were comparably in the two groups. This clinical pathway facilitated trial shows that triclosan impregnation of a 2-0 polydioxanone closing suture can decrease wound infections in patients having a laparotomy for general and abdominal vascular procedures. [54]



The updated SLR included 15 RCTs with 4,800 patients. No publication bias was suggested in the analysis. The predominant effect estimated a relative risk of 0.67 (95% CI: 0.54–0.84,  $p = 0.00053$ ) with an overall lower frequency of SSI in the TS arm than in the NTS arm. Results were robust to sensitivity analysis. The two additional peer-reviewed double-blind RCTs of this update confirmed the predominant effect found in the authors' previous meta-analysis and established the robustness of conclusions that were lacking previously. This SLR and meta-analysis showed that the use of triclosan antimicrobial sutures reduced the incidence of SSI after clean, clean-contaminated, and contaminated surgery. The two additional peer reviewed double blind RCTs reinforced the evidence level of this SLR. [55]

Nakamura and colleagues<sup>2</sup> investigated 410 patients undergoing open and laparoscopic colorectal surgery and showed a non-significant benefit for coated sutures, although a slightly greater reduction in SSI was reported for patients who had open surgery. This non-significant benefit supports Fujita's underlying hypothesis that triclosan-coated are better than uncoated sutures in special high-risk groups, such as in potential wound contamination after open colorectal surgery. [56]

Seven randomized, controlled trials evaluating 1631 patients were retrieved from electronic databases. There were 760 patients in the ABS group

and 871 patients in the simple suture group. There was moderate heterogeneity among trials ( $\tau^2 = 0.12$ ;  $\chi^2 = 8.40$ ,  $df = 6$  [ $P < 0.01$ ];  $I^2 = 29\%$ ). Therefore in the random-effects model, the use of ABS for skin closure in surgical patients was associated with a reduced risk of developing surgical site infections (OR, 0.16; 95% CI, 0.37, 0.99;  $z = 2.02$ ;  $P < 0.04$ ) and postoperative complications (OR, 0.56; 95% CI, 0.32, 0.98  $z = 2.04$ ;  $P = 0.04$ ). The durations of operation and lengths of hospital stay were similar following the use of ABS and SS for skin closure in patients undergoing various surgical procedures. Use of ABS for skin closure in surgical patients is effective in reducing the risk of surgical site infection and postoperative complications. ABS is comparable with SS in terms of length of hospital stay and duration of operation. [57]

Scores for intraoperative handling were favourable and not significantly different for both sutures, although coated polyglactin 910 suture with triclosan received more "excellent" scores (71% vs. 59%). Wound healing characteristics were comparable for both sutures except for pain on postoperative day 1. Significantly fewer patients treated with polyglactin 910 suture with triclosan reported pain on day 1 than patients who received the other suture (68% vs. 89%,  $p = 0.01$ ). The overall incidence of adverse events was 18%; none was device related. Coated polyglactin 910 suture with triclosan performed as well or better than traditional coated polyglactin 910 suture in paediatric patients undergoing general surgical procedures. The incidence of postoperative pain was

significantly less in patients treated with coated polyglactin 910 suture with triclosan than the traditional suture. We speculate that polyglactin 910 suture with triclosan, by inhibiting bacterial colonization of the suture, reduced pain that can be an indicator of "subclinical" infection. Coated polyglactin 910 suture with triclosan may be a useful alternative in patients at increased risk of developing SSI. [58]

The scores for surgeons' evaluation of suture material were favourable and similar for both sutures. Surgeons could not reliably make a distinction in handling between the two sutures. Breaking strength retention was the same for both sutures, ranging from 79% on day 14 to 5% on day 35. Both sutures were essentially absorbed at 70 days post-implantation. Product characterization assessment of the two sutures found them to be indistinguishable. The addition of triclosan to coated polyglactin 910 sutures did not affect physical handling properties or performance characteristics based on the testing and evaluations performed. [59]

The oral LD (50) values for triclosan ranged from 3,750 to 5,000 mg/kg, whereas the LD (50) after subcutaneous injection was >14,600 mg/kg. Safety factors calculated from repeated daily dosing studies ranged from 1,000 to 25,000 times the no-observed-effect levels. There was no evidence of carcinogenic potential in either species, and genotoxicity studies were negative. Reproductive toxicity studies did not reveal any evidence of teratogenic potential. There was

no evidence of skin sensitization potential in controlled studies. Pharmacokinetic studies in animals and humans have shown that triclosan is rapidly absorbed, well distributed in the body, metabolized in the liver, and excreted by the kidneys, with no indication of accumulation over time. Biocompatibility studies showed that coated polyglactin 910 suture with triclosan was non-cytotoxic, non-irritating, and not a chemical pyrogen. In addition, an intramuscular implantation study demonstrated a tissue reaction, a healing response, and an absorption profile comparable to current polyglactin 910 suture. The extensive toxicology database supporting the safety of triclosan and the biocompatibility studies conducted on coated polyglactin 910 suture with triclosan demonstrate the safety of this suture for clinical use. Considering the clinical relevance of surgical site infections and the relatively low level of triclosan required to inhibit bacterial colonization of the suture, the use of this antimicrobial technology is well suited to this application. [60]

A systematic search of both randomized (RCTs) and nonrandomized (non-RCT) studies was performed on PubMed Medline, OVID, EMBASE, and SCOPUS, without restrictions in language and publication type. Random-effects models were utilized and pooled estimates were reported as the relative risk (RR) ratio with 95% confidence interval (CI). Tests for heterogeneity as well as meta-regression, subgroup, and sensitivity analyses were performed.

A meta analysis study included a total of 29 studies (22 RCTs, 7 non-RCTs). The overall RR of acquiring an SSI was 0.65 (95% CI: 0.55–0.77;  $I^2=42.4\%$ ,  $P=.01$ ) in favor of TCS use. The pooled RR was particularly lower for the abdominal surgery group (RR: 0.56; 95% CI: 0.41–0.77) and was robust to sensitivity analysis. Meta-regression analysis revealed that study design, in part, may explain heterogeneity ( $P=.03$ ). The pooled RR subgroup meta-analyses for randomized controlled trials (RCTs) and non-RCTs were 0.74 (95% CI: 0.61–0.89) and 0.53 (95% CI: 0.42–0.66), respectively, both of which favored the use of TCSs. They came to a conclusion that Triclosan coated sutures reduced the risk of SSI by 26% among patients undergoing surgery. This effect was particularly evident among those who underwent abdominal surgery. [61]

Thirteen randomized clinical trials involving 5256 participants were included. Triclosan-coated sutures were associated with lower risk of SSI than uncoated sutures across all surgeries (risk ratio [RR] 0.76, 95% confidence interval [CI] 0.65-0.88,  $P < 0.001$ ). Similar proportions of patients experienced wound dehiscence with either type of suture (RR 0.97, 95% CI 0.49-1.89,  $P = 0.92$ ). Subgroup analysis showed lower risk of SSI with triclosan-coated sutures in abdominal surgeries (RR 0.70, 95% CI 0.50-0.99,  $P = 0.04$ ) and group with prophylactic antibiotic (RR 0.79, 95% CI 0.63-0.99,  $P = 0.04$ ). However, such risk reduction was not observed in cardiac surgeries, breast surgeries, or group without prophylactic antibiotic. They concluded that Triclosan-coated sutures can

decrease the incidence of SSI in abdominal surgeries and might not interfere with wound healing process. Nevertheless, further studies are needed to examine whether triclosan-coated sutures are effective at preventing SSI in non-abdominal surgeries and to further study the interaction of antibiotic prophylaxis with triclosan-coated sutures. [62]

In a study titled Triclosan-coated sutures for the reduction of sternal wound infections? A retrospective observational analysis by Stefan Stadler and Tatjana Fleck, the rate of sternal wound infection was 3.0% in the conventionally closed group, 2.3% in the group with only the sternal fascia closed using triclosan sutures, and 3.2% in the group with total triclosan suture closure (fascia, subcutaneous tissue and skin). They came to a conclusion that Triclosan-coated sutures therefore showed no advantage in avoiding or reducing sternal wound infections. As the cost of these new materials is higher, the rationale for using these sutures remains to be determined.

OBSERVATION

AND RESULT

## OBSERVATION AND RESULT

### STATISTICAL ANALYSIS AND INTERPRETATIONS:

The study subjects were described according to the type variables. The continuous variables were described by averages and the categorical variables were described by percentages. The descriptions of continuous variables were interpreted by student “t” tests and the descriptions of categorical variables were interpreted by  $\chi^2$  tests of goodness of fits. The relationships were analysed and interpreted by  $\chi^2$  tests of independence. The above statistical procedures were performed with the help of the statistical packages namely IBM SPSS statistics-20. The P- values less than or equal to 0.05 ( $P < 0.05$ ) were treated as statistically significant.

### RESULTS:

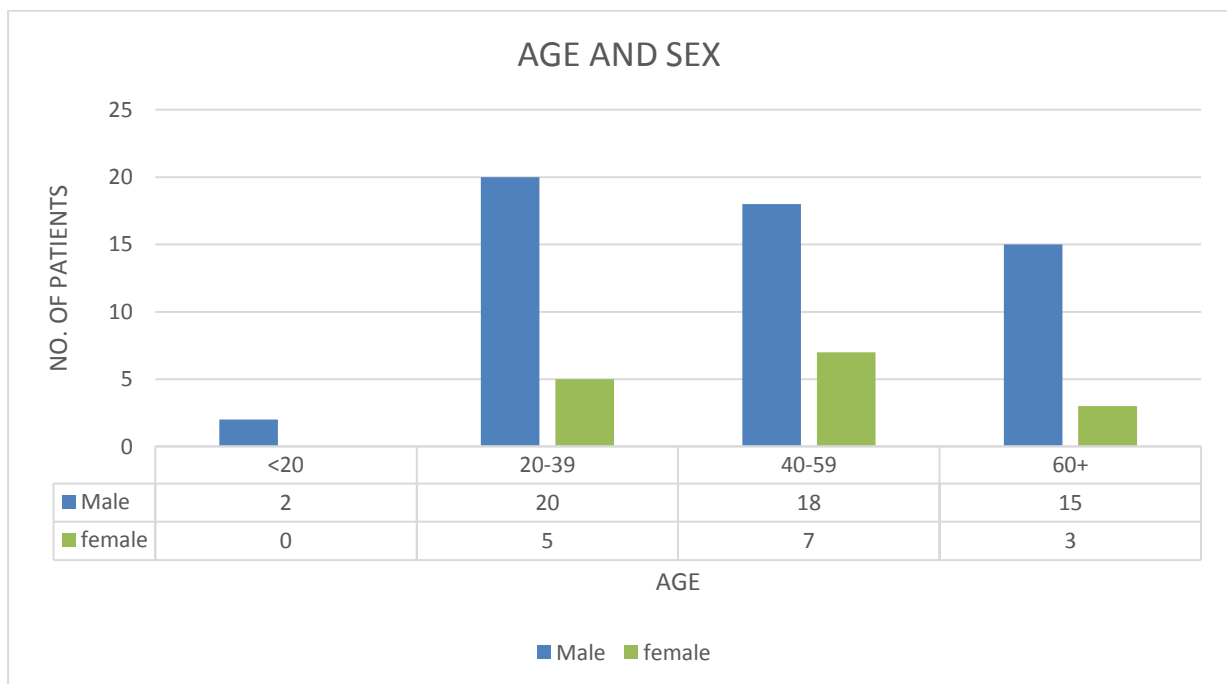
#### DESCRIPTION OF THE STUDY SUBJECTS:

The study subjects namely abdominal surgical cases were described according to their age and gender, type of diagnosis and surgical procedure performed.



Table-1: Percentage distribution of gender wise age group:

Age group (years)	Male		Female		Total	
	Frequency	%	Frequency	%	Frequency	%
<20	2	2.9	0	0.0	2	2.9
20-39	20	28.6	5	7.1	25	35.7
40-59	18	25.7	7	10.0	25	35.7
60+	15	21.4	3	4.3	18	25.7
Total	55	78.6	15	21.4	70	100.0

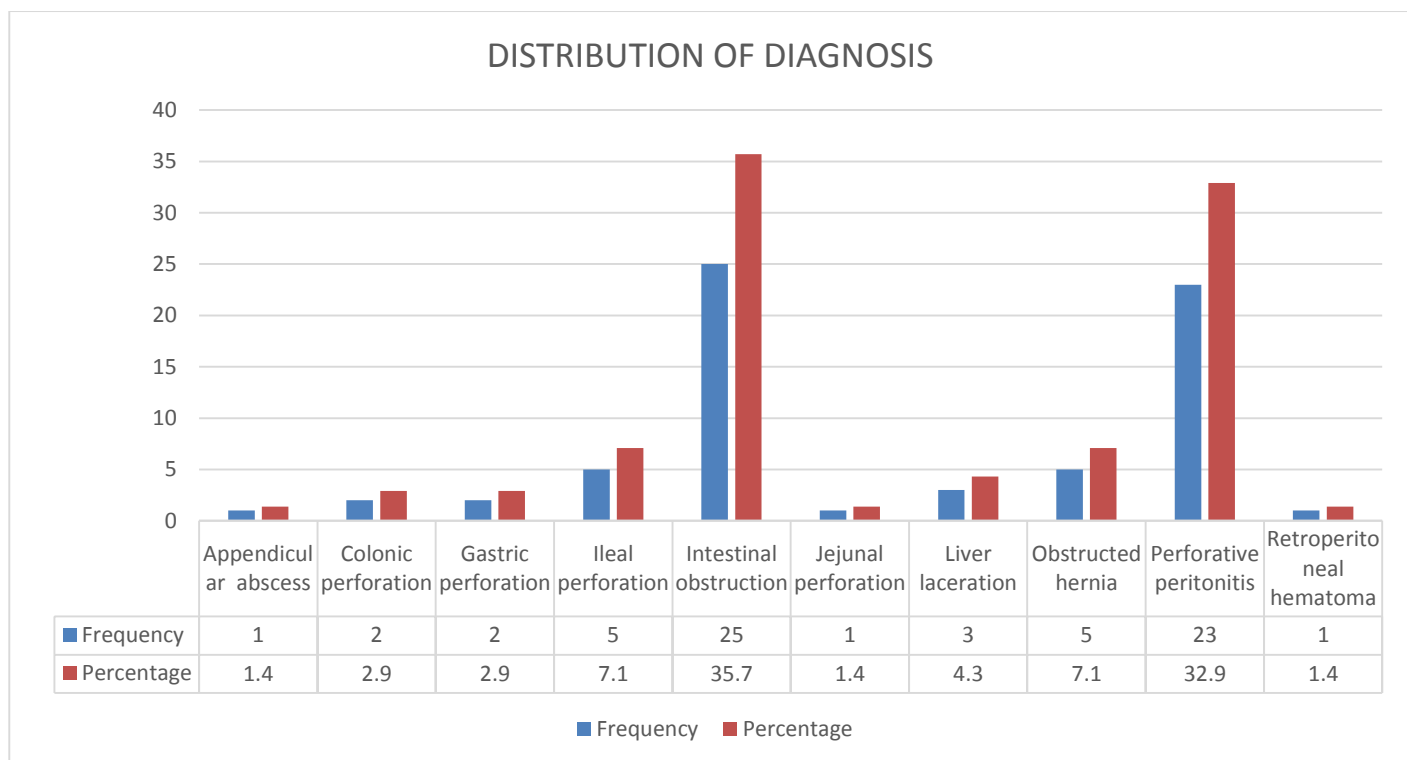


The above table-1 states the age distribution according to the gender of the subjects. The males were 78.6% and females were 21.4%. The mean age of the males was  $44.8 \pm 17.6$  and females were  $50.1 \pm 14.2$  years. The difference between

the mean ages were not statistically significant ( $p>0.05$ ). The total subjects mean age was  $45.9\pm 17.0$  years with a range of 18-86 years.

Table-3: Percentage distribution of Diagnosis.

Sl. No	Diagnosis	Frequency	Percentage
1	Appendicular abscess	1	1.4
2	Colonic perforation	2	2.9
3	Gastric perforation	2	2.9
4	Ileal perforation	5	7.1
5	Intestinal obstruction	25	35.7
6	Jejunal perforation	1	1.4
7	Liver laceration	3	4.3
8	Obstructed hernia	5	7.1
9	Perforative peritonitis	23	32.9
10	Retroperitoneal hematoma	1	1.4
Total		70	100.0



The diagnosis of study subjects were posted in the above table-3. The Intestinal obstruction was the major (35.7%) diagnosis and next was the Perforative peritonitis (32.7%). Both of Appendice abscess (1.4%) and retroperitoneal hematoma (1.4%) was the least diagnosed symptoms.

Table-4: Percentage distribution of Procedures:

Sl. No	Procedures	Frequency	Percentage
1	Adhesiolysis	10	14.3
2	Colostomy	5	7.1
3	Gastrojejunostomy	1	1.4
4	Ileostomy	1	1.4

5	Lavage and drainage	6	8.6
6	Obstruction release	1	1.4
7	Omental patch	23	32.9
8	Primary closure	6	8.6
9	Resection anastomosis	17	24.3
Total		70	100.0

The table-4 states the procedures adopted among the study subjects. The Omental patch (32.9%) was the leading procedures of the subjects. The 24.3% persons had undergone the procedure Resection anastomosis as second most procedures. The Gastrojejunostomy, Ileostomy and Obstruction release were the least scored procedure with 1.4% each. The differences between them were statistically very highly significant ( $P < 0.001$ ).

Table-5: Percentage distribution of Diabetics:

Sl. No	Diabetic status	Frequency	Percentage
1	Diabetic	17	24.3
2	Non-Diabetic	53	75.7
Total		70	100.0

The above table -5 classifies the diabetic status of the study subjects as diabetic and non-diabetic. The diabetic subjects were 24.3% and non-diabetics were 75.7%. The difference was statistically very highly significant ( $P<0.001$ ).

Table-6: Percentage distribution of Wound class:

Sl. No	Wound Class	Frequency	Percentage
1	2	16	22.9
2	3	24	34.3
3	4	30	42.8
Total		70	100.0

The type of wound was stated in the above table-6. The types 2, 3, and 4 were 22.9%, 34.3% and 42.8% respectively. The difference between the wound classes was not statistically significant ( $P>0.05$ ).

Table-7: Association between wound class with SSI.

Wound class	Positive		Negative		Total	
	No	%	No	%	No	%
2	6	8.6	10	14.3	16	22.9
3	7	10.0	17	24.3	24	34.3
4	10	14.3	20	28.5	30	42.8
Total	23	32.9	47	67.1	70	100.0

The wound class was associated with the positive and negative of SSI. The wound class and SSI were not statistically significantly correlated.

Relationships between abdominal closures with suture materials:

The relationships between wound class, 3<sup>rd</sup> day, 7<sup>th</sup> day and total suture infection were studied. The relationship of suture infection was associated with type of culture positive.

Table-8: Association between the suture materials used and infections on 3<sup>rd</sup>

Day of surgery:

Suture materials	-		+		Total		$\chi^2$	df	Sig.
	No	%	No	%	No	%			
V	24	34.3	10	14.3	34	48.6	.945	1	P>0.05
V+	29	41.4	7	10.0	36	51.4			
Total	53	75.7	17	24.3	70	100.0			

The association of suture materials with the outcome of infections on 3<sup>rd</sup> were showed in the above table-8. There was no statistically significant association between the suture materials with outcome of infections on the third day of surgery (P>0.05).

Table-9: Association between the suture materials used and infections on 7<sup>th</sup>

Day of surgery:

Suture materials	-		+		Total		$\chi^2$	df	Sig.
	No	%	No	%	No	%			
V	28	40.0	6	8.6	34	48.6	4.296	1	P<0.05
V+	35	50.0	1	1.4	36	51.4			
Total	63	90.0	7	10.0	70	100.0			

In the above table-9, the relationship between the suture materials with the outcome of infections was stated on the seventh day of operations. The results revealed that there was statistically significant relationship between them ( $P<0.05$ ). The positive outcome of infection (8.6%) of V was significantly differed with the positive outcome of infection (1.4%) of V+. Similarly the negative outcome of infections (40%) was associated with V.

Table-10: Association between the suture materials used and total SSI:

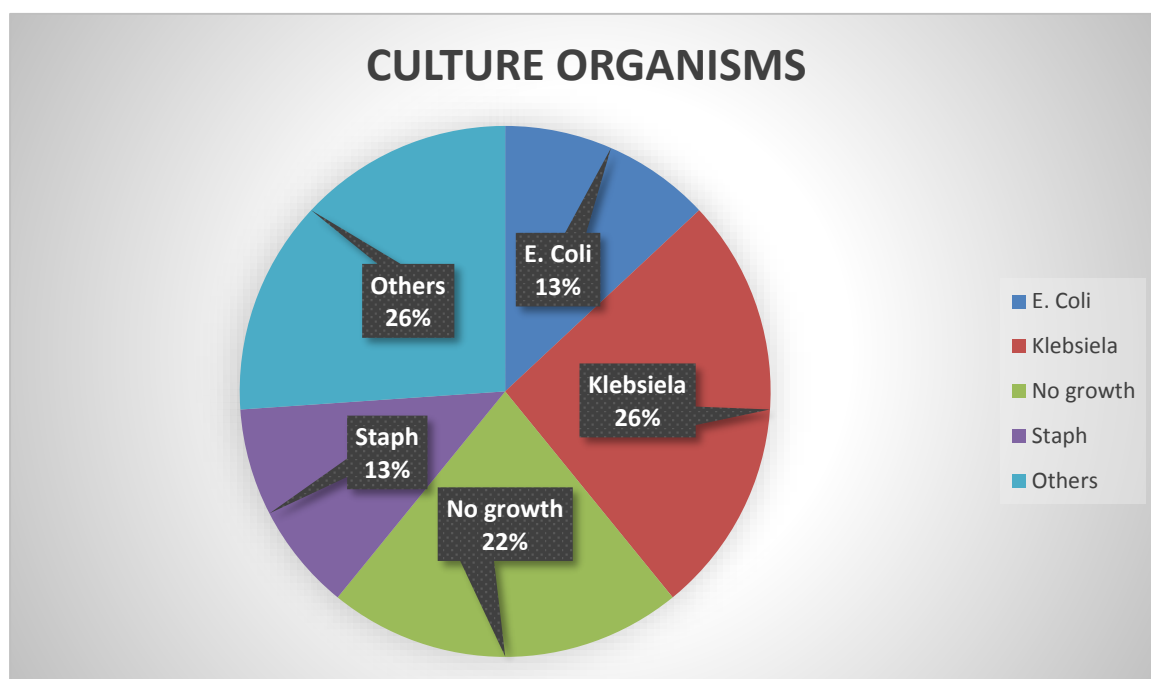
Suture materials	-		+		Total		$\chi^2$	df	Sig.
	No	%	No	%	No	%			
V	19	27.1	15	21.5	34	48.6	3.842	1	P<0.05
V+	28	40.0	8	11.4	36	51.4			
Total	47	67.1	23	32.9	70	100.0			

The above table-9 states the relationship between the suture materials and the total outcome of infections. The results revealed that there was statistically significant relationship between them ( $P < 0.05$ ). The positive outcome of infection (21.5%) of V was significantly differed with the positive outcome of infection (11.4%) of V+. Similarly the negative outcome of infections (27.1%) was associated with V and negative outcome of infections (40.0%) was associated with V+ materials.

Table-11: Percentage distribution of type of Culture positive:

Culture positive	Frequency	%
E. Coli	3	13
Klebsiel	6	26
No growth	5	22
Staph	6	26
Others	3	13
Total	23	100





The above table-11 states the culture types of total SSI. Among them E.Coli was 4.3%, Klebsiel 8.6%, no growth 7.1%, Staph 8.6% and others 4.3%.

# DISCUSSION

## DISCUSSION

Surgical site infection remains a major burden in healthcare and so it is imperative that more research is done to find new innovative ways of reducing it. The purpose of this study was to evaluate the effectiveness of antimicrobial coated suture vicryl plus from Ethicon (triclosan coated polyglactin 910 suture) in reducing superficial surgical site infection in clean wounds.

The contaminated and dirty wound infection rate is mainly associated with incidence of post operative wound infections. It is for this reason that we also decided to study wound infection rates in the emergency setup so as to assess the quality of surgical care we give to our patients. From the study we found the infection rate for clean-contaminated wound to be 21%, which was considerably higher than the expected rate for clean wound which is less than 10%. For contaminated cases it was 33 % while the expected rate was 20%, which is also high. But for dirty cases the incidence was 35 % which was within the expected range of <40%. [8, 10] Therefore, there is certainly need to do more in prevention and management of SSI at our facility.

The age of the patient was not found to be a contributing risk factor in the development of SSI. There was no age group associated with an increased risk of developing SSI (P value 0.761). Our study population had a median age for males was 44.8 and females was 50.1.

From this study we demonstrated a reduction of superficial SSI when triclosan coated polyglactin 910 (vicryl plus) was used as compared to plain vicryl. There was a significant statistically difference (P-value <0.05) demonstrated between the two sutures. This is in line with some previous studies that also demonstrated significant difference between the two sutures. It is important to note that the mechanisms leading to surgical site infections are not fully understood, however the presence of a foreign material like a suture is known to lower the size of bacterial inoculi necessary to develop infection hence creating an antibacterial environment within the wound is supposed to reduce the risk of SSI,. This was the thinking behind the creation of antimicrobial coated suture.

Although vicryl plus has been demonstrated to reduce SSI in some areas like abdominal surgery, it has not been found to be effective in others.

One possibility is that, like all good innovations it may be overused and misused. The widespread use of triclosan for many years in topically personal hygiene products like toothpaste, soap etc. may lead to diminished antimicrobial activity. This inevitable can lead to the development of drug resistance, this has been demonstrated in some studies

The other issue of concern is safety when using triclosan coated sutures, although several studies have demonstrated triclosan to be relatively safe in classic toxicological terms.

Currently in the United States, the Food and Drug Administration (FDA) is reviewing the safety and efficacy of triclosan. And so it would be prudent to exercise caution when using triclosan coated sutures.

# CONCLUSION

## CONCLUSION

In conclusion since there was a definite advantage inferred to the patients by using triclosan coated polyglactin 910, it is the opinion of the researcher that triclosan coated sutures has a role to play in reducing SSI in clean-contaminated, contaminated and dirty wounds and its use should be confined to areas where its application has proven benefits. However more studies should be done to clearly define its role and indications in surgery. Microbiological culture and sensitivity should be done, for all the patients who developed SSI so as to elucidate local causative agents and the most effective drugs. Microbiological testing for local patterns of resistance to triclosan should also be done. Prudent use of antimicrobial so as to reduce the development of drug resistance.

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# ANNEXURE

## **ANNEXURE I**

### **LIST OF FIGURES**

S.NO	TITLE
1	CLASSIFICATION OF SURGICAL SITE INFECTIONS
2	SUPERFICIAL SURGICAL SITE INFECTION
3	DEEP SURGICAL SITE INFECTION
4	SOUTHAMPTON WOUND GRADING
5	TRICLOSAN MOLECULAR STRUCTURE
6	BACTERIAL COLONIES (DARK RED) ARE VISIBLE IN ALL AREAS OF A PETRI DISH EXCEPT FOR THE "ZONE OF INHIBITION" AROUND A ANTIBACTERIAL SUTURE
7	TRICLOSAN COATED SUTURE

## **ANNEXURE II**

### **PROFORMA**

1. Name:
2. Age:
3. Sex:
4. Inpatient no:
5. Comorbidities:
6. Height:
7. Weight:
8. Diagnosis:
9. Class of wound:
10. Procedure done:
11. Suture material used:
12. Adverse effects:

Adverse effect	III POD	VII POD	30 <sup>th</sup> POD
Purulent discharge			
Pain/tenderness			
Localised Swelling			
Redness			
Raised local temperature			

13. CULTURE:

## **ANNEXURE II**

### **CONSENT FORM**

I exercising my free power of choice, hereby give my full free and voluntary consent for myself to be a subject of the study “ABDOMINAL CLOSURE WITH ANTIBACTERIAL COATED SUTURE MATERIALS AND ITS RELATION TO THE INCIDENCE OF POST OPERATIVE SUPERFICIAL SURGICAL SITE INFECTION RATES”.

I have been informed to my satisfaction by attending surgeon Dr. .... the purpose of the study, the clinical and microbiological investigations that are to be carried out and the nature and consequences of the surgery, anaesthesia and the likely complications in my own language.

I am aware of my right to not to opt for this study without having to give reasons for doing so.

Signature of the surgeon:

Signature of the patient:

Date:

Date:



S.NO	NAME	AGE	SEX	IP. NO	DIAGNOSIS	PROCEDURE	DIABETIC	HIV	BMI	CLASS OF WOUND	SUTURE USED	SSI	III POD	VII POD	30TH POD	CULTURE
1	ESAKKIMUTHU	38	M	11131	PERFORATIVE PERITONITIS	OMENTAL PATCH	N	N	26		4	V	-	-	-	
2	MOOKAMMAL	55	F	11777	PERFORATIVE PERITONITIS	OMENTAL PATCH	N	N	32		4	V+	-	-	-	
3	CHELLAPA	62	M	12142	OBSTRUCTED HERNIA	RESECTION ANASTOMOSIS	N	N	22		3	V+	+	-	-	staph
4	KRISHNAN	41	M	12461	PERFORATIVE PERITONITIS	OMENTAL PATCH	N	N	26		4	V	-	-	-	
5	SUDALAIMADAN	35	M	12214	INTESTINAL OBSTRUCTION	RESECTION ANASTOMOSIS	N	N	26		3	V+	-	-	-	
6	GOMATHY	35	F	13079	INTESTINAL OBSTRUCTION	ADHESIOLYSIS	N	N	28		2	V	+	-	-	staph
7	ABDUL NAFOOR	86	M	13159	INTESTINAL OBSTRUCTION	ADHESIOLYSIS	Y	N	21		2	V	+	-	-	klebsiella
8	MAYANDI	60	M	13572	STAB INJURY	PRIMARY CLOSURE	Y	N	32		3	V+	-	-	-	
9	AKBAR ALI	62	M	13619	OBSTRUCTED HERNIA	RESECTION ANASTOMOSIS	N	N	27		3	V+	+	-	-	e. coli
10	MARI	30	M	13835	PERFORATIVE PERITONITIS	OMENTAL PATCH	N	N	31		4	V+	-	-	-	
11	THANGAM	56	F	13830	INTESTINAL OBSTRUCTION	RESECTION ANASTOMOSIS	Y	N	34		3	V+	-	-	-	
12	GUNASEKARAN	20	M	14820	STAB INJURY	PRIMARY CLOSURE	N	N	29		3	V	+	-	+	no growth
13	MAHER NISHA	53	F	15345	OBSTRUCTED HERNIA	OBSTRUCTION RELEASE	Y	N	34		2	V+	-	-	-	
14	SUBRAMANIAN	61	M	15317	PERFORATIVE PERITONITIS	OMENTAL PATCH	N	N	24		4	V+	-	-	-	
15	SUBBULAKSHMI	35	F	15445	INTESTINAL OBSTRUCTION	ADHESIOLYSIS	N	N	32		2	V	+	-	-	others
16	MAHARAJA	24	M	15548	PERFORATIVE PERITONITIS	OMENTAL PATCH	N	N	28		4	V	-	-	-	
17	GANESAN	72	M	16214	PERFORATIVE PERITONITIS	OMENTAL PATCH	Y	N	22		4	V	-	-	-	
18	KANNAN	40	M	16282	PERFORATIVE PERITONITIS	OMENTAL PATCH	N	N	29		4	V	+	-	-	staph
19	KALYANI	55	F	16956	PERFORATIVE PERITONITIS	OMENTAL PATCH	N	N	33		4	V+	-	-	-	
20	MADASAMY	70	M	17496	INTESTINAL OBSTRUCTION	COLOSTOMY	N	N	21		3	V	-	-	-	
21	BALAJI SHANKAR	46	M	18211	APPENDICULAR ABSCESS	LAVAGE AND DRAINAGE	N	N	25		4	V+	+	-	-	klebsiella
22	PON RASATHI	58	F	16288	INTESTINAL OBSTRUCTION	COLOSTOMY	N	N	32		3	V+	-	-	-	
23	RADHA	70	M	18725	INTESTINAL OBSTRUCTION	RESECTION ANASTOMOSIS	Y	N	22		3	V	-	-	-	
24	SURESH	18	M	19477	LIVER LACERATION	LAVAGE AND DRAINAGE	N	N	22		2	V+	-	-	-	
25	BALASUBRAMANIAN	50	M	19702	JEJUNAL PERFORATION	PRIMARY CLOSURE	Y	N	27		3	V+	-	-	-	
26	SAKUNTHALA	33	F	19740	OBSTRUCTED HERNIA	RESECTION ANASTOMOSIS	N	N	34		2	V+	-	-	-	
27	SHANMUGAVEL	65	M	20609	INTESTINAL OBSTRUCTION	ADHESIOLYSIS	Y	N	22		2	V	+	-	+	others
28	CHELLAPOO	63	F	21536	INTESTINAL OBSTRUCTION	ADHESIOLYSIS	N	N	28		2	V+	-	-	-	
29	MANGALASUNDARI	47	F	21637	ILEAL PERFORATION	PRIMARY CLOSURE	N	N	27		3	V+	-	-	-	
30	KRISHNAN	65	M	21863	INTESTINAL OBSTRUCTION	RESECTION ANASTOMOSIS	Y	N	28		3	V	+	-	-	no growth
31	SIVASAKTHIKUMAR	25	M	22122	PERFORATIVE PERITONITIS	OMENTAL PATCH	N	N	22		4	V	-	-	-	
32	AMLOOR PAVAM	80	F	23116	INTESTINAL OBSTRUCTION	ADHESIOLYSIS	N	N	34		2	V+	-	-	-	
33	SUDALAI MUTHU	27	M	23419	COLONIC PERFORATION	COLOSTOMY	N	N	22		4	V	+	-	-	staph
34	GANESAN	45	M	23481	INTESTINAL OBSTRUCTION	RESECTION ANASTOMOSIS	N	N	29		3	V	-	-	-	
35	PETCHIMUTHU	41	M	24108	ILEAL PERFORATION	PRIMARY CLOSURE	Y	N	33		4	V	-	-	-	

36	SIVANKUMAR	76 M	24861	INTESTINAL OBSTRUCTION	ADHESIOLYSIS	Y	N	22	2	V	+	+	-	-	klebsiella
37	NARAYANAN	25 M	25059	INTESTINAL OBSTRUCTION	ADHESIOLYSIS	N	N	26	2	V	-	-	-	-	
38	RAMAR	19 M	25461	COLONIC PERFORATION	COLOSTOMY	N	N	21	4	V	-	-	-	-	
39	AMMACHI	38 F	25571	ILEAL PERFORATION	ILEOSTOMY	N	N	28	3	V	+	-	+	-	no growth
40	MARIAPPAN	25 M	25773	PERFORATIVE PERITONITIS	OMENTAL PATCH	N	N	26	4	V+	-	-	-	-	
41	SAKTHI KANNAN	20 M	27159	INTESTINAL OBSTRUCTION	COLOSTOMY	N	N	29	3	V	-	-	-	-	
42	MURUGESAN	35 M	27552	PERFORATIVE PERITONITIS	OMENTAL PATCH	N	N	21	4	V+	+	+	-	-	e. coli
43	THIMVALI	30 M	27802	LIVER LACERATION	LAVAGE AND DRAINAGE	N	N	31	2	V+	-	-	-	-	
44	BALASUBRAMANIAN	53 M	28075	INTESTINAL OBSTRUCTION	RESECTION ANASTOMOSIS	N	N	34	3	V	-	-	-	-	
45	KARTHIKA SELVI	28 F	28168	INTESTINAL OBSTRUCTION	RESECTION ANASTOMOSIS	N	N	29	3	V	+	+	-	-	others
46	PERUMAL	70 M	28360	PERFORATIVE PERITONITIS	OMENTAL PATCH	Y	N	22	4	V	-	-	-	-	
47	PALAVESAM	25 M	28548	GASTRIC PERFORATION	OMENTAL PATCH	N	N	27	4	V	-	-	-	-	
48	ARUMUGAM	24 M	28985	INTESTINAL OBSTRUCTION	ADHESIOLYSIS	N	N	26	2	V+	+	+	-	-	staph
49	THANGARAJ	52 M	29924	LIVER LACERATION	LAVAGE AND DRAINAGE	N	N	32	2	V	-	-	-	-	
50	VELU	45 M	30113	INTESTINAL OBSTRUCTION	RESECTION ANASTOMOSIS	N	N	29	3	V	-	-	-	-	
51	PARAMASIVAM	24 M	30349	PERFORATIVE PERITONITIS	OMENTAL PATCH	N	N	21	4	V	+	+	-	-	klebsiella
52	SEETHARAM	50 F	31196	PERFORATIVE PERITONITIS	OMENTAL PATCH	Y	N	32	4	V	-	-	-	-	
53	DURAPALLAM	72 M	31496	INTESTINAL OBSTRUCTION	ADHESIOLYSIS	Y	N	23	2	V	-	-	-	-	
54	PARVATHI	65 F	30542	INTESTINAL OBSTRUCTION	RESECTION ANASTOMOSIS	N	N	34	3	V+	+	+	-	-	e. coli
55	VEERAPANDIAN	38 M	32079	RETROPERITONEAL HEMATOMA	LAVAGE AND DRAINAGE	N	N	26	2	V	-	-	-	-	
56	AYYAPILLAI	47 M	32222	PERFORATIVE PERITONITIS	OMENTAL PATCH	N	N	25	4	V+	-	-	-	-	
57	VENKATARAM	56 M	31160	GASTRIC PERFORATION	GASTROJEJUNOSTOMY	N	N	25	4	V+	+	+	-	-	no growth
58	AYYAKUTTY	62 M	32990	INTESTINAL OBSTRUCTION	RESECTION ANASTOMOSIS	Y	N	29	3	V+	-	-	-	-	
59	HARIHARASUBHU	43 M	33645	PERFORATIVE PERITONITIS	OMENTAL PATCH	N	N	23	4	V	-	-	-	-	
60	KRISHNAN	65 M	35448	PERFORATIVE PERITONITIS	OMENTAL PATCH	N	N	32	4	V	+	+	-	-	staph
61	AAYIRAM	40 M	35766	PERFORATIVE PERITONITIS	LAVAGE AND DRAINAGE	N	N	27	4	V+	-	-	-	-	
62	BALAMURUGAN	27 M	39170	ILEAL PERFORATION	PRIMARY CLOSURE	N	N	24	3	V+	-	-	-	-	
63	VELSAMY	36 M	39229	PERFORATIVE PERITONITIS	OMENTAL PATCH	N	N	28	4	V+	+	+	-	-	staph
64	KOLAPPAN	37 M	45442	PERFORATIVE PERITONITIS	OMENTAL PATCH	N	N	25	4	V+	-	-	-	-	
65	SUDALAIMUTHU	55 M	49640	INTESTINAL OBSTRUCTION	RESECTION ANASTOMOSIS	N	N	34	3	V+	-	-	-	-	
66	RASUKUTTI	50 M	49710	PERFORATIVE PERITONITIS	OMENTAL PATCH	N	N	24	4	V+	+	+	-	-	klebsiella
67	PITCHAIAH	45 M	52621	OBSTRUCTED HERNIA	RESECTION ANASTOMOSIS	N	N	29	3	V+	-	-	-	-	
68	ANAND KUMAR	20 M	54598	PERFORATIVE PERITONITIS	OMENTAL PATCH	N	N	21	4	V+	-	-	-	-	
69	ALAGURAJ	52 M	54651	ILEAL PERFORATION	RESECTION ANASTOMOSIS	Y	N	27	4	V+	+	+	-	-	klebsiella
70	ANNADURAI	43 M	55351	INTESTINAL OBSTRUCTION	RESECTION ANASTOMOSIS	Y	N	21	3	V+	-	-	-	-	

## **KEY TO MASTER CHART**

M – MALE

F – FEMALE

Y – YES

N – NO

BMI – BODY MASS INDEX

+ - POSITIVE

- - NEGATIVE

V – VICRYL

V+ - VICRYL PLUS